Approved March 24, 1992
Date

MINUTES OF THE HOUSE COMMITTEE ON PENSIONS, INVESTMENTS & BENEFITS

The meeting was called to order by Representative Don Rezac at
Chairperson

12:40 a.m./p.m. on March 4, 1992 in room 521-S of the Capitol.

All members were present except: Representative Elaine Wells (excused)
Representative Barbara Allen (excused)
Representative Walker Hendrix (excused)
Representative Tom Love (excused)

Committee staff present:

Alan Conroy - Legislative Research
Richard Ryan - Legislative Research
Gordon Self - Revisor's Office
Juanita Blasdel - Committee Secretary

Conferees appearing before the committee:
Jerry Marlatt - Kansas State Firefighters Assoc.
James Todd - Ks. State Firefighters Assoc.
Meredith Williams - KPERS
Pat Winterringer - Kansas City
Representative Bill Reardon
Charles Rentfro - Kansas City
Ed Marcotte - retired state employee
Others attending: see attached sheet

The meeting was called to order by Chairman Rezac at 12:40 p.m.

Richard Ryan of Legislative Research was called on to give a brief on HB 2598 which was to be heard.

HB 2598 - KP&F, service connected death and disability

The first proponent to speak was Jerry Marlatt of Kansas State Council of Fire Fighters. This bill would simply include cancer as "service connected" with regard to a death or any physical or mental disability. In mortality studies made, it appears that fire fighters have an increased risk of several types of cancer, including cancer of the brain, rectum and colon, skin and leukemia (Attachment #1).

James Todd also of Kansas State Firefighters Association then spoke a few words and asked for support of this bill for the Kansas City, Kansas firefighters. Representative Wisdom asked Mr. Todd to make a statement as to why the Kansas City, Kansas firefighters were not here in support of this bill today. Mr. Todd told the committee that the mother of one of their friends had died and they all went to the funeral today.

Meredith Williams of KPERS then explained a little as to what the bill entailed and the fiscal impact. The cost to the state would be $44,000 per year.

HB 2961 - KPERS, benefits for spouse of deceased member

Hearings were opened on this bill and the first proponent was a widow from Kansas City, Kansas, Pat Winterringer. She spoke from a handout (Attachment #2) and stressed that it was wrong to exclude the wife who had been married to the employee for many years, then divorced, and received none of his pension when he died. She felt the pension had been given to women who did not earn it, but ignored the ones who did.

Meredith Williams of KPERS then told that the bill would eliminate the current restriction wherein the surviving spouse of a deceased member of a police or fire system loses the survivor's benefit during any period of remarriage. This bill would allow the surviving spouse to remarry without losing the benefit. However, he felt there should be a start date.

The chairman then called on Alan Conroy of Legislative Research to give a brief on HB 2970 for hearings.

HB 2970 - KPERS, relating to disability benefits

Representative Bill Reardon was then called on to testify on this bill that he had drafted. Representative Reardon spoke from a handout (Attachment #3). He said this amendment is necessary to assure that disabled persons are not penalized for attempting, in good faith, to return to work after being disabled from an injury or sickness. Under this bill, if a worker returns to work but is later unable to continue due to the earlier disability, they are not penalized for the period they are unable to work. The waiting period does not start over unless it is a different disability that causes the loss of work.

Charles Rentfro of Kansas City, Kansas then told of his experience concerning this 180 day waiting period. He had made a good faith effort to return to work and under KPERS he had to start his 180 days again when he was unable to remain working. He would not benefit from this bill if it were to be passed, because he has already taken his disability.

Meredith Williams of KPERS spoke a few words concerning this bill and said they would administer to the best of their ability if the bill was passed.

Questions were then asked of Mr. Williams.

The chairman asked Alan Conroy of Legislative Research to clarify whether KPERS does or does not compare with social security as to the disability (180 days) and let the committee know.

Chairman Rezac called on Alan Conroy of Legislative Research to give a brief on the next bill to be heard, HB 3162.

HB 3162 - KPERS, relating to benefits thereunder; retirement; participating service

Ed Marcotte, a retired State of Kansas employee, was the first to testify on this bill. He had previously handed out some material "State of Kansas Employee Alert!" (Attachment #4).

Chairman Rezac announced there would be another meeting at noon tomorrow, Thursday, March 5, and again at 5:00 if not finished with the bills.

Meeting adjourned at 1:15 p.m.

GUEST LIST

COMMITTEE: Pensions, Investments & Benefits Date: 3-4-92

Name (Please Print)	COMPANY ORGANIZATION	ADDRESS
J A Jack	KSTA	Wichita
Joe Thibodeau	Ks. St. Firefighters'	Lawrence
Craig Grant	KNEA	Topeka
Robert Linder	KDHE	Topeka
James E Fry	KDHE	Topeka
Gary Cathcart	KRT	Topeka
Arthur Schuman	KDHE	TOPEKA
Richard Stucky	KAPE	Topeka
Keith Haxton	KAPE	Topeka
RICHARD LONG	BD of AG	TOPEKA
GARRY ZIEGEL	KDHE	TOPEKA
IVAN MALCOM	KDOT	Topeka
BILL WEBER	K.D.O.T	TOPEKA.
Jack Fluke	KDOT	Topeka
L.D.(Dean) Strickland	Third Judicial Dist. Court Services	TOPEKA
PAT O'Rourke	KSTA	TOPEKA
Dan Plantz	KDOT	Topeka

Name	Company or Organization	Address
Jerry Scott	KHP	Topeka.
Ed Mirault	~~Division of Water~~ SELF	Topeka
HAROLD GIBBON	Self	Topeka
Pat Winteringer	K.C.Ks. P.D.	Bonner Springs, Ks.
Robert Kibby	Bd. of Agri. Div. Water Res.	Topeka

"PROGRESS THROUGH UNITY"

KANSAS STATE COUNCIL OF FIRE FIGHTERS

Affiliated With
INTERNATIONAL ASSOCIATION OF FIRE FIGHTERS • KANSAS STATE FEDERATION OF LABOR • CENTRAL LABOR BODIES

DATE: March 4, 1992

TO: Members of House Pensions, Investment and Benefits

FROM: Jerry Marlatt, Lobbyist
Kansas State Council of Fire Fighters

RE: House Bill 2598

We strongly support the favorable passage of House Bill 2598. This bill would simply include cancer as "Service Connected" with regard to a death or any physical or mental disability.

Several mortality studies have been performed in fire fighters. When combining these studies, it appears that fire fighters have an increased risk (or incidence) of several types of cancer, including cancer of the brain, rectum and colon, skin and leukemia.

Thank you for your consideration of House Bill 2598.

Pensions, Investments & Benefits
Attachment #1
3-4-92

THE PAGE

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CAREERS IN ASHES

A lot of firefighters are dying of cancer. The burning question: Did they get it on the job?

BY PAUL RUBIN

photo by Jon Gipe; inset photo by Suzanne Starr (reprinted with permission of the Arizona R...)

They found what they were looking for only after rummaging through dusty cardboard boxes at a warehouse on Phoenix's west side. One man held a flashlight so another could read from an old Phoenix Fire Department logbook.

There it was: Call No. 14692, sixteen handwritten lines describing a nitric acid spill in South Phoenix on the evening of April 21, 1977. Firefighters from four stations had responded to clean up the 75-gallon spill. Among them had been David Sanchez and Dale Brandt.

The search started after a reporter wondered whether the department's personnel records on Brandt and Sanchez would reveal any job-related injuries. Injury reports had shown Brandt and Sanchez had been treated that April evening for inhaling the fumes of nitric acid. But those reports didn't explain what happened. The long-buried log found in the warehouse was the only source of those details. Neither Brandt nor Sanchez remembers the incident, which didn't even merit news coverage.

The only reason to search for information on the spill is that Brandt is suffering from terminal brain cancer and Sanchez recently has battled testicular and lymphatic cancer.

Firefighters in Phoenix and around the country are dying of cancer at a rate that seems out of whack compared with the rest of us. Preliminary studies—none of them done yet in Arizona—point to that alarming fact. They also indicate that the various cancers that have struck firefighters may be linked to the chemicals they've inhaled, touched or swallowed during years of battling blazes.

This silent chemical assault on firefighters—historically beloved, larger-than-life figures who save lives and property—only now is being recognized. The search for a link is just starting. And it's going to be difficult.

Did cancer smite Brandt and Sanchez because they were exposed to nitric acid in April 1977? Nitric acid is known to cause respiratory problems, but it's not necessarily a carcinogen. There's no proof that their cancers are connected to that acid spill. They and other firefighters have spent years eating smoke and slogging through all sorts of spilled chemicals. The 1977 spill may help pinpoint an exposure to something that led to cancer. It just as likely may be a dead end.

"Don't remember it," Brandt says with a short, sarcastic laugh, referring to the 1977 spill. "That's like asking me if I remember going to the bathroom on a certain date."

Phoenix firefighters talk about being surrounded by smoke that literally made their testicles tingle. Was that smoke cancer-causing? Fire departments everywhere, including Phoenix, have been late to recognize the dangers in today's chemical environment. And their records are sketchy. Yet many occupational-health experts say there likely is a link between frequent exposure to chemicals and cancer in firefighters.

A test case is about to happen in Arizona. Firefighter Gary Pykare is preparing to file a claim—unprecedented in this state—that he got cancer on the job and deserves worker compensation. Four states (but not Arizona) have laws that "presume" a link between firefighters and cancer when exposures to toxic chemicals can reasonably be documented. Municipal officials naturally resist that notion because of the money cities might have to shell out to stricken employees.

No one knows the exact number of current and retired firefighters who have contracted cancer, or how they got it. Even Phoenix's department, highly respected within the profession, often doesn't record the exposures of its 973 firefighters to possibly toxic chemicals.

At least eleven Phoenix firefighters in the 1980s have either died from cancer or have contracted it. No one can say if Phoenix's firefighters or those at other Arizona departments have a higher incidence rate of cancer than the general public. That's because no one in the state ever has studied cancer in firefighters.

Studies done in Los Angeles, Seattle, New York City, and Toronto tend to show a much higher rate of cancer in firefighters than in the rest of the population. None of those studies, however, is considered definitive, and scientists cannot answer the key question: Are firefighters getting cancer from battling blazes and spills?

Phoenix Fire Captain David Sanchez, whose cancer went into remission a few months ago, is certain of it. "It's hard for me to believe that I didn't get cancer from the job," he says. "Car fires, house

"They gave me six months, and that was six months ago," says firefighter Dale Brandt, who is dying of brain cancer. Brandt rests from his work at a blaze on Bell Road in June 1987, before his illness struck (inset above): poses in firefighting gear (right); cradles his niece, Michelle, last month (above).

Attachment #1-2

FIREFIGHTERS

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of Arizona, an umbrella group for the state's fire unions. "We have good soldiers who are thinking twice for the first time. You knew you could fall through a roof or anything, but when the danger was over, it was over. You don't know where it ends anymore."

The Arizona State Legislature appropriated $100,000 this June to try to determine how widespread cancer is in Arizona's firefighters. Only nine of ninety legislators opposed funding the bill, which Governor Rose Mofford signed into law.

One of those opposing it was New River Republican Gary Giordano. "I don't want to be perceived as being in favor of cancer for firefighters, but I don't think it's a necessary expenditure," says Giordano. "We had just passed a bill to set up a procedure to track cancer through the Department of Health Services, that does everything that this study is supposed to do. The firefighters' union wants total control over the study, rather than handing it over, for better or worse, to the bureaucrats at DHS."

Giordano is confused. It is, after all, a new subject for Arizona. He's referring to a $192,000 appropriation last session that forces hospitals to release cancer and birth-defects statistics to a DHS computer registry. The idea is to allow health officials to pinpoint potential clusters as they turn up. The new cancer-registry law, sparked by fear of cancer in Maryvale, does not provide for epidemiological studies, which try to define and explain factors that may determine how and why a group of people get a disease.

The cancer-registry law and the study of cancer in firefighters were two separate bills and "are two separate issues," says Dr. Timothy Flood, a DHS doctor. He is overseeing the cancer registry, but he knows about the issue of cancer in firefighters.

"We know that firefighters run across an alphabet soup of chemicals and combustibles during the course of their work," Flood says. "The problem is, there just are no definite studies out there for anyone to cite. This will be a start."

The doctor who likely will head the firefighter-cancer committee knows the task will be difficult—the hundred grand is enough only for a preliminary gathering of statistics. "We have a sleuthing job of the first magnitude on our hands—a creative, scientific sleuthing job," says Dr. James Schamadan, an occupational-health specialist who was the DHS director in 1974-75. A former staff physician for the Phoenix Fire Department, Schamadan is chief executive officer of Scottsdale Memorial Hospital system.

The committee's job will be thorny, says Bruce Teele, a fire-service specialist for the Boston-area-based National Fire Protection Association, because of the lack of data. "Very few agencies can say, 'This is what's happening to us,' because they don't keep proper track of what's happening," Teele says. "Even in Phoenix, where you have, in my opinion, the premier chief in the country, I'd bet the stats aren't there." They aren't.

BRUNACINI'S 1985 textbook *Fire Command* is a model how-to, according to national fire officials. His department is regarded nationally as among the best in safety and management practices.

"Bruno," as everyone calls him, was a blood-and-guts type during his years on the truck. He still longs to fight fires. "Every minute of my life," he says. "When my wife, Rita, hears a siren, she goes, 'They're playing our song.'"

Fire Captain David Sanchez is back at work after battling cancer.

The Brunacinis' two sons, Nick and John, are Phoenix firefighters. The chief knows how hard it is to make his sons and other young, gung ho firefighters strictly follow new safety procedures. He agrees, however, that even if firefighters are painstakingly cautious, it's still unclear how much protection clothing and equipment provide in the long run from toxic chemicals.

"We're just beginning to understand this stuff," Brunacini says. "The young guys say, 'What about you, look how you did it.'

"Those old, leather-lunged firemen used to really ride you."

I say, 'That was then, this is now, and you're going to be careful and follow the rules, period.' Firefighters have always been full of piss and vinegar. That's how you survive. We don't think, we feel. When you start fooling around with the way people feel, you've got problems. And firefighters have always been bullheaded by nature."

If firefighters are the same, the fires they fight aren't. The reason is chemicals. About 500 new compounds go into commercial use each year, according to government reports, and many end up where there are fires. Burning buildings are being seen more and more as toxic predators, and firefighters face exposure to chemicals by inhaling, ingesting or absorbing them through the skin.

Scientists say they don't know exactly what happens when chemicals burn in fires. Even a living-room couch with its plastic covering can be hazardous to firefighters during or after a blaze. Fires involving plastics, solvents, PCBs, pesticides, asbestos routinely emit known or suspected carcinogens.

"Over a lifetime, the cumulative effect of those low and high exposures will place firefighters at an increased risk for work-related illnesses," says Scott Barnhart, associate director of the Occupational Medicine Program at the University of Washington. "The range and toxicity of exposures is likely to increase. Studies to identify these hazardous exposures are going to be difficult."

In the early 1970s, the scuba-type, Darth Vader-looking masks called SCBAs replaced the canister masks that firefighters had worn for decades. The old masks were designed to filter out carbon monoxide, but little else.

Veteran Phoenix firemen—until 1979, the department was all male—resisted the SCBAs and ridiculed the younger firemen who wore them. "It was a badge of honor not to wear one," recalls David Sanchez. "Those old, leather-lunged firemen used to really ride you. Meanwhile, companies were making new chemicals, making a 'better' place for us to live in, without knowing the long-term effects on the person who breathed them."

In the last few years, the Phoenix department has stiffened its safety rules. These days, firefighters are required to keep their breathing units on during overhaul—the cleanup time after the flames are out when smoke and soot swirl around them.

Firefighter and union activist Mike Bielecki warns, however, "The SCBA is not infallible, it's not a panacea. It's a help, but you can't see at night when you wear it, so you pull it off. Or it gets pushed off your face on the fireground. Boom. Cancer."

ONE IN THREE Los Angeles firefighters are expected to develop cancer by the age of sixty, compared with one in five people in general. A study, completed in 1982 by the Institute of Cancer and Blood Research, showed incidence rates in Los Angeles firefighters of up to three times above normal for mouth or throat cancer, and more than twice above normal for cancers of the brain, lung, rectum, pancreas and prostate. Cancer-related deaths among Los Angeles firefighters doubled from 17 percent in 1950 to 36 percent in 1980.

The Los Angeles project is recognized by many occupational-health experts as one of the better analyses of the relationship between firefighters and cancer. Still, Dr. Howard Bierman, who headed the study, emphasized, "The mortality study represents only the first phase of a multiphase study. Further research is needed if we are to perceive the total cancer picture for city firefighters."

Bierman noted the difficulties of trying to estimate mortality trends, especially when using death certificates. "Cancer victims may even be certified as having died of some totally different cause," Bierman wrote. "Even if cancer is correctly diagnosed while the patient is alive, the relevant information may never reach the death certificate." The effect of those and other statistical distortions, Bierman warned, was to underestimate the true number of cancer deaths in Los Angeles firefighters. (Six years later, the rest of the Los Angeles research has not been completed.)

"We are now in the new fire environment," says Irving Selikof, professor emeritus at New York City's Mount Sinai School of Medicine. "We have all the old chemicals. We have no knowledge what happens when they are in fire. We have the new chemicals, of which we know very little. We have a whole new set of problems."

Problems like the one Phoenix firefighters had in May 1986 during a blaze at a west-side metal-plating factory. After the three-alarmer was contained, several firefighters—including members of the HAZMAT team—went inside to extinguish it.

One firefighter noticed that his protective overcoat was bubbling—not from heat, but apparently from a reaction to the chemical stew brewing at the still-smoky factory. Several other firefighters at the site felt an eerie tingling in their testicles. They hightailed it outside.

The long-term outlook for many firefighters is as unpredictable as that smoldering factory fire. "I've treated guys

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FIREFIGHTERS

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firefighters had sent in more than 11,000 reports of exposures—about 40 percent of them at residential fires.

The Phoenix department has patterned its own chemical-exposure record form after the one in California. Veteran firefighters like Gary Pykare, however, are lucky to have any record of exposures in their files. Still, Pykare plans to push his worker-compensation claim.

Pykare's lawyers asked two cancer experts in mid-June if they would look at his case. Dr. Stanley Leong of the Tucson-based Arizona Cancer Center and Dr. Melissa McDiarmid, a Johns Hopkins University professor of occupational medicine, agreed separately last month to take a look.

Pykare will have a year to file his claim after the doctors issue their opinions. His attorneys estimate that it may take one or two years after that to resolve matters.

Pykare says he won't wait to see if Arizona's lawmakers enact a cancer-presumption law. "It's time to put up or shut up," Pykare says. "You never know what the legislature is going to do, anyway. Time to force the issue."

SOMEONE SEES a tramp spread-eagled on the pavement at Seventh Street and Missouri and phones for help. David

"Don't ever trust smoke. It can hide what's really going on, spread the fire, burn, blow up and really ruin your life—sometimes almost at once."

Sanchez, a 34-year-old Phoenix fire captain, responds with a small squad of paramedics.

"What did you take today? Cocaine?" Sanchez asks the tramp.

"Some heroin and some booze," the man finally answers. "Anything to kill the pain. I got cancer."

"What kind of cancer?"

"I don't know. They just cut all that shit off me."

Sanchez decides to have an ambulance take the man to the county hospital for a once-over. A half-hour later, back at Station 17—a homey-looking firehouse at the corner of 16th Street and Missouri—Sanchez takes a breather from the scorching July day.

"Who knows if he really has cancer?" Sanchez wonders. "Poor guy has enough problems without that."

Sanchez is back at work full-time after dueling with testicular and lymphatic cancer. His stamina isn't back to where it was, but it's getting there.

During the next seven hours, the firefighters respond to ten calls: There are car accidents, heart attacks, slips-and-falls, everything but a fire. At one call, a 59-year-old man's heart stops for about thirty terrifying seconds. Under Sanchez's direction, the firefighters bring the man back from the almost-dead and speed him to a hospital.

Life-saving stuff, but not what most firefighters long for. "They would almost sell their souls for a fire," Sanchez says. "Not in terms of trying to wish ill of people, but it's fun to fight fires."

Since his fight with cancer, some of his firehouse bravado and gallows humor is gone. "It makes you reconsider your own mortality," he says. "When you're seventeen, eighteen, nineteen, you do a lot of crazy things—you perceive that you've got a long time ahead of you. But you don't. We see a lot of stuff—guts spattered all over the [illegible] dead children. You're close to de[illegible]t's normal. You tend to become callous. That's nature's way of protecting the organism."

Sanchez, a 1971 Bourgade Catholic High School graduate, joined the Phoenix department after a short stretch as a police trainee. "People are much more amiable to a fireman than to cops," he says. "We're just not perceived the same way. We're good guys."

Sanchez became a medic in 1976, but, like most other firefighters, he never much considered his own health. "I was right there when the department became more cognizant of safety, but we were lax," he recalls. "I used to have the mentality to run into buildings, put that fire out, let the chips fall where they may. We were quick to pull off our masks. At car fires, we'd pull them right off. After just about any fire, we'd blow out snot for the next few days."

Last November, Sanchez felt a lump in a testicle. It took him several weeks to get to a doctor—"I knew what the possibilities were, the repercussions, and it just took me time to go in." Tests showed that he had cancer that already was attacking his lymph glands and chest.

"Every time I've heard of lymph cancer, the prognosis has been pretty grave," he says, "but what I had was exquisitely sensitive to chemotherapy, and it was in an early enough stage."

Sanchez underwent chemotherapy this year from January through April. "You're the worst sick you've ever been for three or four days," he says of the treatments. "It's like being hung over, beaten up and the worst flu you've ever had. You can see that people who have to do it for a long, long time sometimes say, 'No more. Let me die.' But when I felt better, I'd go to work."

Now, he says, he's changed his approach to firefighting.

"If there's no life-threatening danger to a citizen," he says, "why should we expose ourselves to something that could get us down the road? Why not just let that car burn if no one's in it? Unless the risk is calculable, there's no reason to subject yourself to a death sentence. This is a career, a good living, but it's a job. When you leave, you're not going to get a day like Martin Luther King. It's just a job."

Chief Brunacini agrees that firefighters face a dilemma: How far do you go to save property when your actions could give you cancer? In his book, *Fire Command*, Brunacini devotes a chapter to the "axioms" of firefighting.

"A lot of the stuff we risked our lives for on Saturday night," he writes, "gets loaded into an old dump truck and hauled off Monday morning." Another axiom goes, "Safe firefighters are smart firefighters. Vomiting firefighters are ugly firefighters."

And, finally, "Don't ever trust smoke. It can hide what's really going on, spread the fire, burn, blow up and really ruin your life—sometimes almost at once."

An alarm at the station interrupts Sanchez. A teen-ager at a church has taken a karate chop to the head and needs medical help. As the firefighters rush out, Sanchez holds back a second to make a point. He's changed, but he still has a firefighter's wry sense of humor.

"A lot of guys don't know how to approach me," he says. "They don't know how to say 'cancer' in front of you. They want to know your prognosis without asking you. When I was sick, I finally told them, 'If you want to do something to help me, open up my chest and take this stuff out.'"

IAFF DEPARTMENT OF OCCUPATIONAL HEALTH AND SAFETY

SUMMARY OF THE VIEWS ON OCCUPATIONAL CANCER IN FIRE FIGHTERS

Thank you for requesting information on Occupational Cancer in Fire Fighters. This document will serve to summarize and interpret the contents of the informational packet enclosed, as well as briefly state the views of the IAFF Department of Occupational Health and Safety on this matter.

I. Summary of Cancer in Fire Fighters

(1) It has been documented in scientific studies that fire fighters are exposed to thousands of different chemical agents during the course of their duties. Many **industrial hygiene** studies performed in fire fighters have actually measured exposures at real and simulated fires.

(2) Some of these chemicals are known to be carcinogens (cancer-causing agents). Most of the studies that have suggested that certain chemicals can cause cancer have been performed in animals, but some human epidemiologic studies do exist.

(3) Some of the chemicals to which fire fighters are exposed have been documented in epidemiologic studies to increase the risk of cancer in working populations (such as workers manufacturing or applying the agent). These include **vinyl chloride, asbestos, benzene, and polycyclic aromatic hydrocarbons (tars)**. These have been shown to cause liver cancer, lung and lung lining cancer, leukemia, and skin and lung cancer, respectively. These studies have not been performed in fire fighters, however.

(4) Several **mortality studies** have been performed in fire fighters (some of the reports are enclosed in this packet). When combining these studies, it appears that fire fighters have an increased risk (or incidence) of several types of cancer, including cancer of the **brain, rectum and colon, skin, and leukemia.** Other cancers, such as bladder cancer, have been found to be elevated in some studies but there is a lack of consistency in the findings.

The position of the IAFF Department of Occupational Health and Safety is that there is an increased incidence of some specific cancers in fire fighters.

(5) The information in the preceding paragraph may seem alarming but should be tempered with some additional knowledge. First, brain cancer and leukemia are uncommon cancers and an increased incidence of an uncommon cancer still results in relatively few cases of cancer. Secondly, rectum and colon cancer and skin cancer can be **cured** if detected and treated **early**. Finally, fire fighting will not cause any of these cancers if certain precautions to prevent exposures on the job are taken (for example, conscientious use of SCBA). Job-related cancers, like all occupational diseases, can be **prevented**.

(6) An interesting type of cancer that is surprisingly missing from the above list is **lung cancer**. A logical question is: Why don't fire fighters, who are exposed to chemicals in fire smoke identical to the lung-cancer-causing chemicals in cigarette smoke (called polycyclic aromatic hydrocarbons, or PAHs), have an increased incidence of lung cancer? This is a difficult question to answer. Fire fighter mortality studies have not found a convincing increased risk of lung cancer. This may be due to problems with the design or conduct of the studies.

(7) The issue of increased cancer in fire fighters is not without controversy, however. Some scientists feel the data are not clear; this is because of lack of consistency in the studies in their conclusions and the use of different methods and definitions. There are many epidemiologic reasons for this lack of agreement, so the IAFF Department of Occupational Health and Safety concludes that the **body of the evidence** weighs in favor of an increased incidence of certain cancers in fire fighters.

II. Definitions of Some Common Terms in Cancer Epidemiologic Research

(1) **Healthy worker effect:** In mortality studies, the incidence rate of a certain cause of death is **compared** to the incidence rate in some general population (usually the entire U.S. population). As fire fighters are healthier as a whole than the U.S. population, with certainly much less heart and lung disease (as these conditions would preclude the person from becoming a fire fighter), when rates of death from some cause in fire fighters are compared to the general rates fire fighters look to have a much decreased risk of the cause of death. This apparent decreased risk may not have been found if the fire fighters had been compared to a working population with similar health requirements (such as police). The decrease is thus an **epidemiologic artifact** that has been called the healthy worker effect.

(2) **Incidence rate:** This is the number of cases of disease in a population divided by the number of persons in the population at risk for the disease in a specified period of time (usually a year). The passage of time is an important requirement here.

(3) **Mortality study:** In fire fighter mortality studies, the causes of death are counted up for the fire fighters (the **observed** number of deaths) then compared to the **expected** number of deaths in the fire fighters if they had the **same rate of death** as some comparison population (usually the general U.S. population). These studies are performed in **cohorts** of fire fighters, some defined population of fire fighters with the criteria for study specified in advance (such as a minimum number of years employed, a certain city, etc.).

(4) **PMR (proportionate mortality ratio):** This is one common measure of the effect of fire fighting (or other jobs or exposures) on the incidence of disease in a fire fighter mortality study. The PMR looks at all the deaths in the population of fire fighters and calculates the **percent** (or **proportion**) of deaths due to a specific cause (for example, 35% of deaths were due to heart disease). This percent is then divided by the percent of deaths due to a specific cause in a comparison population. **This ratio is the PMR.** It is usually then multiplied by 100 so that PMRs above 100 mean "increased risk" (for example, a PMR of 270 is interpreted to mean that fire fighters had 2.7 times the risk of a certain cause of death). In general, the PMR is not thought to be as good an estimate of the risk of death due to a job or exposure as the SMR. PMRs are subject to many potential problems which often make them less valid epidemiologic tools.

(5) **Polycyclic aromatic hydrocarbons (PAHs):** Polycyclic means "many rings" (the molecular structure is in a ring or circle shape); aromatic means "similar to **benzene** in molecular structure"; and hydrocarbons means that the molecule consists of the atoms hydrogen and carbon. These chemicals, also known as tars, are known human carcinogens.

(6) **Risk:** Risk is expressed as a number between 0 and 1 (and if multiplied by 100 gives "**percent**"). It is most relevant for fire fighters in the context of cancer when expressed as the lifetime risk of developing a certain kind of cancer (for example, over the lifetime of a typical fire fighter, there is a 14% risk or **chance** of cancer "X"). The risk of a certain cancer in fire fighters can be divided by the risk of the same cancer in a comparison population to give a ratio of risks. If this is above 1, then there is an increased risk of this cancer in fire fighters.

(7) SMR (standardized mortality ratio): When the **observed** number of deaths from a mortality study (see above) is divided by the **expected** number of deaths based on the death rates in a comparison population, this ratio is called an SMR. The term "**standardized**" usually means that the effects of age (because cancer is known to increase with age, if one population is older than the other it would have an increased number of cancer deaths for this reason) have been removed by adjusting or standardizing the ages of the two populations (the two populations are the fire fighters and the comparison population).

III. Contents of Packet

Scientific Articles

Note: These articles may be difficult for lay people to read and understand completely.

1) Bureau of Health Statistics, Research and Evaluation, Massachusetts Department of Public Health. Cancer Incidence Among Massachusetts Firefighters 1982-1986. March 1990.

This study examined cancer incidence in fire fighters, compared to the police and the State as a whole. It found significant excess of melanoma, bladder cancer, and non-Hodgkin's lymphoma. Fire fighters were also found to have excess pancreatic cancer and leukemia when compared to the police. The findings of this study are consistent with other studies of fire fighter cancer incidence and adds weight to the existing body of evidence that fire fighters are at increased risk of developing occupationally induce cancers.

2) Heyer N. Cohort Mortality study of Seattle Fire Fighters, 1945-1983. February 1988.

Particularly noteworthy is the summary of fire fighter cancer mortality studies on pages 16-19 and the results compared to other studies on pages 41-44.

3) Vena JE, Fiedler RC. Mortality of a municipal-worker cohort: IV. Fire Fighters. Am J Industrial Medicine 1987; 11: 671-684.

This is one of the better studies in the field. It is somewhat difficult to read, but armed with the knowledge that an SMR above 100 means "increased risk" of that cancer, the abstract (before the introduction on the first page) is useful to read.

4) Rosenstock L, Demers P, Heyer N, Barnhart S. Northwest fire fighter mortality, 1945-1983. September 1987.

This document has a summary on page 1 and an excellent review of the topic of cancer in fire fighters on pages 4-9.

5) Feuer E, Rosenman K. Mortality in police and fire fighters in New Jersey. Am J Industrial Medicine 1986; 9: 517-527.

This is a study that measured "PMRs" which are generally thought to be less scientifically useful than SMRs. Its strength is that it compared fire fighter mortality to police mortality, a better comparison (these two groups are more similar except for their exposures) than fire fighters with

the general population. This addresses the issue of the "healthy worker effect."

6) Lewis SS, Bierman HR, Faith MR. Cancer mortality among Los Angeles City Fire Fighters. February 1983.

This is a reasonably easy to read and contains good discussions of problems interpreting these types of studies (pages 6-7) and mortality patterns (pages 8-9).

IAFF Pamphlets

1) Occupational Cancer and the Fire Fighter. Department of Occupational Health and Safety, IAFF, 1982.

This is a useful summary of the most important issues in easily understood terms. It is strongest from the perspective of exposures.

Articles from the Lay Press

1) "Deadly Smoke", by Chuck Cook and Marla Cone. The Register, Santa Anna, CA, December 1983.

This is an emotional account of illness in fire fighters.

2) "Careers in Ashes", by Paul Rubin. New Times, Phoenix, AZ, August 1988.

Another emotional account of cancer in fire fighters which includes discussions of many important topics in the area.

OCCUPATIONAL CANCER AND THE FIRE FIGHTER

DEPARTMENT OF RESEARCH
HEALTH AND SAFETY DIVISION

INTERNATIONAL ASSOCIATION OF FIRE FIGHTERS
AFL-CIO-CLC

Attachment #1-10

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Attachment #1-11

INTRODUCTION

Of all the diseases that strike the human body, cancer provokes the most fear. Cancer comes from the Greek word, karkinos, meaning crab. It is a term that applies to numerous diseases that can afflict the human body, each of which has its own history and characteristics. Cancers are classified according to their appearance under a microscope. More than one hundred different types of cancer have been identified.

No one is immune: athletes, movie stars, political leaders, children. It affects one of every four people. In the United States, someone dies of cancer once every two minutes. It is estimated that almost three-quarters of a million people will develop cancer annually in the United States alone; approximately 400,000 will die from the disease. In addition, approximately 40,000 Canadians died of cancer representing almost one-fifth of all deaths in Canada. Many of the major forms of cancer such as lung, intestine, stomach and bladder can be associated with occupational exposure.

Cancer can strike any age group, but it is most commonly found among the middle-aged and the elderly. Likewise, the sex of an individual can play a role in determining the type of cancer contracted. In men, the lung, prostate and large intestine are among the most common organs stricken with cancer. Cancers of the breast, uterus and the large intestine are those most frequently encountered in women.

There is also a difference among countries as to the most common types of cancer. For example, stomach cancer is much more common in Japan than it is in the United States and Canada. Likewise, liver cancer is much more common in Africa. The differences in the rate of certain cancers is probably linked to the differences in lifestyle, environment and occupations.

This manual reviews (1) how our body's cells normally function; (2) what causes cancer; (3) how carcinogens are identified; (4) how cancer is medically treated; (5) the relationship between cancer and occupation; (6) the relationship between cancer and fire fighting; and (7) guidelines for determining whether cancer is occupationally related.

HOW CELLS FUNCTION

The human body is made up of billions of cells. Cells, which are tiny structures of different shapes, make up the tissues of the body as well as carry out all of the body's functional activities. Even though the functions of cells of the various body organs differ, the reproduction of all cells is the same. That is, all cells reproduce by dividing. Cell growth and replacement goes on continuously in all living tissues.

There are three major types of tissue in the human body: static tissue, expanding tissue and renewing tissue. Once a person reaches normal adult size, static cells lose their capacity to divide. Static cells are found in muscle and nerve cells, thus if you lose muscle or nerve cells they cannot be replaced. Expanding cells also stop growing when the body reaches its normal adult size, but they retain the capacity to divide again if parts of the tissue is removed. For example, if half of your liver is removed, then your liver still has the capacity to grow back to its original size. However, once the liver reaches its regular size again, the cells know that they must stop dividing. The third type of cells are known as renewing cells, since they continually renew cells lost on a daily basis. For example, white blood cells have a lifespan of only five or six days in the bloodstream before they are replaced.

The process of replacing cells is performed in an orderly fashion that corresponds directly to the kind and amount of cells that require replacement. During normal cell growth, the parent cells duplicate themselves so that the resulting daughter cells are identical. For example, cells in the skin when they divide develop into other skin cells. In the human body, there exists an equilibrium between the death of worn-out cells and the growth of new cells.

Each and every cell contains a genetic substance known as DNA (deoxyribonucleic acid) which acts as the control center to direct the cell's functions. If there is a change in the structure of DNA, then the cell will not function normally. When cancer is involved, the change in DNA produces uncontrollable cell division that can frequently lead to a mass of tissue known as a tumor. Cancer cells apparently will continue to grow regardless of the size of the mass created. Once cancer attacks an organ, such as the lungs, the essential functions of that organ are gradually destroyed. The cancer victim also becomes more susceptible to infection or heart failure, which often is responsible for death. These masses of tissues known as tumors can be either benign (not cancerous) or malignant (cancer-
s).

Benign tumors are those that are self-contained in an organ or tissue of the body. These tumors can become life threatening when they cause bleeding, crowding or interfere with the functions of vital organs. Although they can grow very large, when removed surgically, benign tumors usually do not grow back. Certain types of benign tumors do represent the early stages of malignant tumor development.

Malignant tumors are those that separate and travel through the blood stream or lymph channels to other parts of the body to form satellite tumors. These types of tumors can travel into normal tissue and yet retain their own characteristics, continue abnormal growth and continue to send out other "seed" cells to form still more satellite tumors. For example, many cancer cells can break into blood vessels and be carried to other parts of the body such as the lungs or the liver. Eventually, these cancer cells will divide to produce another cancer. This process of cancer spreading from one part of the body to another is known as metastases. In some instances, even though the original tumor is removed, the disease may return because the cancer had spread to other regions of the body.

WHAT CAUSES CANCER

There are a variety of factors that appear to influence the susceptibility of an individual getting cancer including age, smoking, diet, air pollution, exposure to industrial chemicals, variations in the body's natural immune defense system, use of medication and infection with viruses.

There are also some factors that are known not to cause cancer, such as an injury. Bumps and bruises cannot cause the development of cancer. Another false conception without scientific validation is that cancer is contagious. You cannot get cancer by coming into contact with a person afflicted with the disease.

From the medical research performed thus far, it also does not appear that cancer can be inherited. Although data are available which shows that members of the same family may develop the same type of cancer (particularly of the breast, stomach, colon, prostate, lung or uterus), scientists have not yet determined whether this phenomenon is caused by inherited characteristics or by similar lifestyles within a family. Thus, what may appear to be inherited cancer may in fact be a case of family members having similar lifestyle patterns.

The interaction of chemicals in the human body that results in cancer is another puzzle that scientists have yet to solve. However, there is agreement regarding certain pieces of that puzzle.

It is generally believed that cancer is caused through a two-stage process: initiation and promotion. In some instances, a single chemical performs both roles. Sometimes it is the interaction of several chemicals that produces the cancer.

In this two-stage theory, a chemical or chemicals initiates genetic changes in the body's cells that are irreversible. This action usually occurs very quickly. A cell can remain in this initiation stage indefinitely or it can be stimulated by a substance or substances to promote the formation of full cancer cells. The promotion stage can occur immediately after the initiation stage or even months later and still cause cancerous tumors to be produced. According to this theory, the production of cancer cells can only occur if the initiator substance attacks before exposure to the promoter substance.

How such initiated cells are actually promoted to tumors is not yet known. However, it is probable that promoters infiltrate our bodies through our diets and the environment and affect those cells that have been initiated. Thus, this two-stage cycle may explain why there is a higher incidence of lung cancer among smokers exposed to asbestos than what would be expected from exposure from either the substances or the additive (synergistic) effect of the two substances.

These promoters and/or initiators are known as carcinogens. A carcinogen is a cancer-causing agent that enters the body through the skin, through the lungs when breathing or through the digestive system if they are eaten. There are two types of carcinogens, direct acting and indirect acting. A direct acting carcinogen usually causes cancer at the site of the exposure. Indirect acting carcinogens are chemically changed after entering the body. For example, benzidine compounds can enter the body through the skin or lung but do not cause skin or lung cancer. Once in the body, these benzidine compounds are then chemically changed into substances called metabolites which are then eliminated from the body in the urine. When these metabolites pass through the urinary bladder, they can cause changes in the lining which can result in bladder tumors or bladder cancer.

A complete carcinogen is a chemical that acts as both an initiator and a promoter. Exposure to small doses of such chemicals will cause them to initiate tumor formation. At high levels of exposure, these complete carcinogens will both initiate and promote the production of full cancer cells.

The length of time it takes to develop cancer differs depending upon which carcinogen(s) the person was sufficiently exposed to. This characteristic is known as a "latent" effect. In fact, the average time lapse to develop chemically induced cancer is about 20 years. One of the problems of linking cancers to occupational exposures is the long period of time that frequently passes between an exposure to a carcinogen and the development of the cancer. The length of time it takes to develop cancer after exposure to a carcinogen appears to be due to the fact that a cell must pass through several stages before it becomes a malignant cancer cell.

LATENCY PERIOD

Carcinogen	*Time Elapsed From Initial Exposure*
Arsenic	30-50 years
Asbestos	20-40 years
Chromium	10-20 years
Nickel	15-30 years

Combinations of certain agents can also produce a "synergistic" effect. That is, the effect of the combination of two or more agents is greater than what would otherwise be expected from the sum of the individual components. This effect has been found among two known carcinogenic agents as well as the combination of a known carcinogen with a normally noncarcinogenic agent.

Carcinogens are also known to cause many other adverse health effects including dermatitis, chemical skin burns, eye and skin irritation, damage to the lungs and other organs, and so forth. These

alth effects are usually acute. That is, they occur immediately or soon after an exposure to a high concentration of a carcinogen. In addition, some carcinogens can also have mutagenic or teratogenic effects. A mutagen causes a change in the genes that can be passed on from generation to generation, while a teratogen causes direct injury to the embryo or fetus.

IDENTIFYING CARCINOGENS

Since the beginning of World War II, the production of synthetic chemicals has increased 350-fold. With the addition of thousands of new chemicals annually, it becomes impossible to study the carcinogenic properties of each and every one of them. The 1980 National Institute for Occupational Safety and Health (NIOSH) *Registry of Toxic Effects of Chemical Substances* contains more than 45,000 substances, which have been shown to produce some type of toxic effect on animals and/or humans. NIOSH estimates that there are approximately 100,000 substances that may have some type of toxic effects. Of the substances listed in the *Registry* almost 3,000 have demonstrated a positive relationship to carcinogenicity.

Carcinogens include synthetic and natural chemicals, radiation from isotopes, x-rays and sunlight and certain viruses. Synthetic chemicals can be used for industrial purposes, food or cosmetic additives, pesticides or pharmaceuticals. Actually, in proportion, relatively few substances are known to cause cancer. Most chemicals, even those that are toxic or dangerous are not carcinogenic. However, since cancer takes an average 20 years to develop in humans after the exposure to a carcinogen, it is difficult to determine exactly what agent may have caused the disease.

The Occupational Safety and Health Administration (OSHA) considers a chemical to be a carcinogen if a properly designed study shows exposure to that chemical has caused cancer in:

humans,

two different animal (mammal) species,

one animal species if the results are duplicated in separate studies,

one animal species if the results are supported by multi-test evidence of mutagenicity (short-term tests).

There is no simple method to detect a carcinogen. One frequently used method is the epidemiologic study. Epidemiology has been defined as the study of the origin, nature, pathology and prevention of diseases temporarily prevalent in a community or throughout a large area. Generally, epidemiological cancer studies examine mortality rates retrospectively (historically). In a retrospective epidemiologic study, workers who have contracted cancer could be looked at to determine if the cancer is more prevalent among persons exposed to certain chemicals. To make such a determination, the group of workers being studied could be carefully correlated with a control group with similar demographic characteristics (e.g., age, sex, smoking habits).

For example, to conduct a retrospective epidemiological study of the carcinogenic effect of vinyl chloride, NIOSH selected four plants

that had been polymerizing vinyl chloride for between 20 and 32 years. NIOSH then reviewed the medical records of workers who had been working with vinyl chloride for at least 5 years and had their first exposure at least 10 years ago. This NIOSH study found higher-than-expected rates of cancer deaths of the brain and central nervous system, respiratory system, liver, lymph nodes and blood-forming tissues. NIOSH also found that death rates were correlated with the length of exposure; the longer the exposure, the higher the death rate.

Another common type of epidemiological study is conducted prospectively. That is, the study could look at workers with known exposure to suspected or known carcinogenic agents, but who have not yet developed cancer. The eventual health effects of the exposed population could then be compared with an unexposed population or control group. While a prospective study is the only method that can definitely assess the cause-effect relationship of exposure to a substance and the onset of disease, it is extremely time-consuming and costly.

Both retrospective and prospective human epidemiology provides post facto data regarding the health effects of certain chemicals. That is, they gather information from a population that has already been exposed to the substance. However, it is the most reliable way to assess the risk of the substance on the human population as well as a vital method to determine any adverse health effects. Epidemiologic studies do have many drawbacks to contend with.

- Epidemiologic studies are not effective at disease prevention, especially for cancers which have latency periods of up to 40 years. In such cases, it may take many years to determine whether a substance is indeed carcinogenic while workers are still being exposed. Retrospective epidemiologic studies also cannot assess the impact of new substances being added into the environment.
- The long latency period of cancer makes it difficult and expensive to study human populations.
- Since people are exposed to a variety of substances at differing amounts in their job, home, diet and so forth, it may be extremely difficult to single out one causal factor. This is especially true for cancer because of the possibility of synergistic effects and the long latency period.
- It is difficult to establish a control group, a large portion of the population that has little or no exposure to the substance.
- The population under study must be carefully selected to avoid the inclusion of persons not exposed to the substance being studied.
- Individuals are often hesitant to participate, which may make obtaining the necessary medical records difficult if not impossible.

When these population studies have been conducted, they have resulted in the identification of at least 30 carcinogenic agents.

In laboratory testing, mice or rats are generally used because they are small, easy to handle and more economical than larger animals. Mice and rats also appear to react in a manner similar to humans when exposed to carcinogens. In fact, most of the major forms of human cancer have been duplicated in the laboratory using such animals. Since the natural lifetime of rodents is only two to three years, they can provide data about the cancer-causing capabilities of test materials more quickly than longer living animals like dogs or monkeys. Special strains of rodents have also been developed to specifically test for carcinogenic agents.

Almost all of the 30 carcinogenic agents that have been identified with population studies have also been shown to cause cancer in laboratory animals. What is not known is how many of the several hundred chemicals that have been shown to cause cancer in laboratory animals may also cause cancer in humans. However, research has shown that materials which are found to cause cancer in one mammalian species are usually found to cause cancer in other species as well. For example, chemicals such as vinyl chloride were shown to cause cancer in mice prior to documenting their carcinogenic effects in humans.

Thus, it is generally assumed that agents that are found to be cancer-causing in laboratory animals will have a similar effect on humans.

Laboratory tests are performed using about 50 rodents of each sex and exposing them to different doses of the testing material for a period of about two years. Other groups are treated in the same fashion, but are not exposed to the test substance to set up a control for the experiment. The exposed group often receives high dosages of the chemical, so that any potential carcinogenic effects are more likely to be detected when working with a small group of rodents. Likewise, when large dosages do not cause the development of cancer, there is more assurance that the test substance is not a carcinogen. Research guidelines do restrict dosage levels to prevent premature deaths of the animals, since the animals must live long enough for the tumor to develop. Finally, the laboratory rodents are dissected and examined by pathologists (doctors who can relate changes in body tissue to disease causal factors). A test substance is a carcinogen when the number of tumors found in the exposed group is higher than that found in the control group.

While high doses of many chemicals are toxic, they are not neces-

sarily carcinogenic. Toxic chemicals may cause loss of hair or weight, various organ malfunctions and even death without causing cancer to develop. During one study, 120 pesticides and industrial chemicals were given to mice at the highest dosage levels they could tolerate and still survive. Even though these chemicals were specifically selected for their suspected carcinogenicity, after two years of such treatment only 11 of these chemicals caused cancer in the test mice.

The use of high doses in these tests does not mean that low dosages will not cause cancer. For example, a carcinogenic agent may cause cancer in one out of every 10,000 subjects exposed to it. While this may seem like a very small amount, when we consider the exposure of 220 million people, we would have a result of 22,000 cancers—a public health disaster. Thus, in order to identify the carcinogen that may cause cancer in one out of every 10,000 subjects, you would need to test thousands of rodents which would be extremely time-consuming and expensive. By exposing the rodents to a dose 5,000 times greater than might actually cause cancer, then about 50% or about 20-30 of the test group might develop cancer. If this rate is higher than the control group, then it can probably be concluded that the test chemical is carcinogenic.

It should also be noted that even though one species may develop cancer under certain conditions while another does not, the positive result in any adequately performed test does indicate a cancer risk for humans.

Animal studies to determine whether a substance is carcinogenic are both time-consuming and expensive. Often times such tests require as much as two years to test one chemical in a single species at a cost of more than $200,000. Even if the funds were available, there is not enough laboratory space or trained scientists to test every chemical for carcinogenicity.

Thus, new short-term tests are being developed to provide preliminary results of the carcinogenicity of chemical agents. In these tests, various types of cells growing in laboratory cultures are treated with the test material to determine whether subsequent growth is normal or abnormal. One of the most popular of these tests is known as the *Ames Salmonella* mutagensis system, or simply, the Ames test. Early evidence indicates that almost 90% of all chemicals that are shown to be carcinogenic in human/animal studies also are found to give positive results (abnormal growth) in these short-term tests. Presently, no short-term tests are used as the basis for determining whether a chemical will be carcinogenic in humans.

There are a variety of lists indicating known or suspected carcinos published by the federal government and other organizations. ppendix I.)

Known carcinogens are usually defined as those substances associated with cancer in humans. Suspected carcinogens are those substances that have shown sufficient evidence of cancer causation in animals and are reasonably believed to also cause cancer in humans. A list of all known or suspected carcinogens to which a significant number of persons are exposed is prepared annually by the National Toxicology Program, U.S. Public Health Service for publication by the Secretary of the Department of Health and Human Services. The Environmental Protection Agency's Carcinogen Assessment Group compiles another list of carcinogens. The National Cancer Institute, the Occupational Safety and Health Administration and the National Institute for Occupational Safety and Health are also among the governmental agencies that publish information on known or suspected carcinogens. The International Agency for Research on Cancer is another organization that evaluates substances for their carcinogenicity.

NON-OCCUPATIONAL RISK FACTORS

There are many things that we choose to do in our daily lives that can reduce or increase the risk of getting cancer.

Smoking—People who smoke will develop lung cancer 10 times more often than nonsmokers. Smokers are likewise more prone to develop cancers of the throat, mouth, esophagus, pancreas and bladder. The increased risk of smoking is compounded in occupations, such as fire fighting, where exposure from asbestos dust occurs; though this factor has frequently been ignored by those in the fire service. Those who choose fire fighting as a profession must also realize that cigarette smoking will increase their risk of developing cancer. This is not a scare tactic, simply the fact.

Drinking—Heavy drinking of alcoholic beverages increases the risk of cancers of the esophagus, mouth, throat, larynx and liver. The risk of getting cancer is magnified when a person combines heavy drinking and smoking.

X-rays—Such examinations are frequently crucial for a physician to make the proper diagnosis and conduct treatment, however, the routine use of X-rays should be avoided unless specifically prescribed by your physician. X-rays taken during pregnancy increases the chances that the child will develop leukemia.

Solvents—Long-term exposure to solvents such as found in household and industrial cleaners, cleaning fluids and paint thinners may be hazardous if inhaled in high concentrations.

Eating habits—Although there is no clear evidence that eating habits influence the risk of getting cancer, the research conducted thus far does suggest that the risk of cancer is reduced for those who t less fatty foods and more high-fiber foods such as bran, whole grains and fibrous fruits and vegetables. There is also evidence that overweight people have a higher probability of getting some types of cancer, especially breast cancer, than slim people.

DIAGNOSIS AND TREATMENT OF CANCER

Many forms of cancer are curable, especially when found in an early stage prior to the tumor spreading to other parts of the body. However, it is also likely that at any early stage, symptoms have yet to develop. Thus, periodic examinations should be performed by your physician. Routine medical examinations can often detect cancers of the breast, cervix, prostate, colon and rectum prior to the appearance of any symptoms.

While cancer does not show any symptoms in the beginning stages, symptoms may appear before the disease spreads.

There are seven warning signs of cancer.

1. Unusual bleeding or discharge.
2. A lump or thickening in the breast or elsewhere.
3. A sore that does not heal.
4. Change in bladder or bowel habits.
5. Hoarseness or cough.
6. Indigestion or difficulty swallowing.
7. Change in a wart or mole.

If any of these signs persist for more than two weeks, then a physician should be consulted. It should also be remembered that the number of symptoms that can be present far exceeds this list. Likewise, the absence of all of these signals does not necessarily mean that cancer is not present. These symptoms are merely indications that medical attention is required as soon as possible. It should also be noted that pain is not an early warning sign of cancer.

Diagnosing cancer is difficult because each type of cancer has different characteristics. Thus, diagnostic procedures are usually determined by the individual's complaints and the physician's suspicions. Although there are various methods available to detect the presence of a tumor, the determination of whether the tumor is benign or malignant is performed through a procedure known as a biopsy. This procedure involves the removal of a small piece of tissue from the tumor which is examined under a microscope for the presence of cancer cells.

If cancer is diagnosed, treatment should be immediately initiated in a medical facility that has the staff and resources to administer the type of treatment you may require. There are three main methods of treating cancer: surgery, radiation therapy (radiotherapy) and chemotherapy (drug therapy). For some types of cancer, a combination of these methods may prove to be the most effective means of treatment.

Surgery is the primary method of cancer treatment, especially if the disease is detected in an early stage. Basically, cancer surgery involves the removal of the tumor and nearby tissue that may contain cancer cells. Almost half of all treated cancer involves surgery.

Radiation therapy uses X-rays or radiation from radioactive substances such as cobalt or radium. This type of therapy is one of the primary methods for treating cancers of the bladder, cervix, skin and parts of the head and neck.

Some cancers do not respond to radiation at all and are called radio-resistant. These cancers, which must be treated by surgery, include the gastrointestinal tract, brain tumors, cancers of the breast, kidney, testis and ovary, and bone and muscle. Those cancers that do respond to radiation are known as radiosensitive cancers. These types of cancers are often treated by a combination of radiation and chemotherapy. Such cancers arise from the lymph nodes and blood-forming tissues.

Surgery and radiation therapy both share similar characteristics and limitations in treating cancer patients. Both are based on treating a localized area and usually involve destroying normal cells in order to kill cancer cells. The principal advantage of radiation therapy is that it can avoid the need for disfiguring surgery. The major disadvantage to radiation therapy is that damage to normal tissue can also be fatal.

Chemotherapy is the most recent form of cancer treatment. Currently, there are more than 50 anticancer drugs being used. This form of treatment has become increasingly effective because of the ability of anticancer drugs to circulate throughout the body and attack cancer cells that have already spread to other organs.

THE RELATIONSHIP BETWEEN OCCUPATION AND CANCER

In 1775, Percivall Pott, an English surgeon, observed the first documented case of occupational cancer. He attributed the development of cancer of the scrotum in chimney sweeps to their exposure and contact with soot. While Pott discovered the relationship between occupational exposure and cancer more than 200 years ago, coke-oven workers in the steel industry still die of lung cancer at 10 times the rate of other steelworkers because of their exposure to the same kinds of substances that Pott had shown caused cancer in chimney sweeps.

Scientists at the International Agency for Research on Cancer have estimated, based on studies from around the world, that up to 6% of all cancers can be directly related to exposure at the workplace. The National Institutes of Health scientists have concluded that at least 20% of all cancers will be related to workplace exposure. The World Health Organization has estimated that between 75% and 85% of all cancers are caused by environmental exposures.

In the United States, there are about 45,000 chemicals currently in production. Obviously, some of these chemicals are capable of inducing chronic health effects in humans. The occurrence of chronic diseases has an extraordinary large impact on health in the United States and Canada. NIOSH has estimated that the United States has at least 100,000 deaths a year that are directly related to occupational exposures. In addition, probably more than 400,000 new cases of occupationally related diseases are occurring annually.

The cost of chronic diseases, such as cancer, is also staggering. The General Accounting Office has estimated the cost of cancer at $15 billion per year. This estimate was based on the cost of treatment and the loss of earning power and productivity. If social costs (i.e., the costs of pyschosocial deteriorations brought on by a disease but which are not reflected in economic cost analysis) are included then the price tag for cancer may rise to as much as $150 billion annually. The ever increasing cost for medical care only means that these figures are bound to rise even more in the coming years.

There is a wide range of opinion among scientists regarding how much exposure a person can have to a carcinogen to cause cancer. Some believe that a single asbestos fiber could cause a cancerous growth to begin in the lungs. Others believe that exposure to vinyl chloride will not cause cancer until it reacts and uses up all of a noncellular substance that is produced and secreted in the body. Thus, a worker may be able to be exposed to a certain threshold level of vinyl chloride without using up all of this noncellular substance.

Although both opinions may be valid, there are no known scientific methods for determining threshold levels for carcinogens, even

if such thresholds do exist. The American Conference of Governmental Industrial Hygienists has developed threshold limit values for over 400 substances. These values are based on information gathered from industrial experience as well as human and animal studies and represents what is believed to be a level that all workers can be exposed to day after day without adverse health effects.

SELECTED KNOWN OR SUSPECTED INDUSTRIAL CARCINOGENIC AGENTS

Substance	*Target Organ*	*Route of Entry*
Acrylonitrile	lung, colon	inhalation, skin
4-Aminobiphenyl	bladder	inhalation, oral
Arsenic Compounds	skin, lung	oral, inhalation
Asbestos	lung and chest cavity gastrointestinal tract	inhalation, oral
Auramine	bladder	oral, inhalation, skin
Benzene	bone marrow	inhalation, skin
Benzidine	bladder	inhalation, oral, skin
Beryllium Compounds	lung	inhalation
Bis(chloromethyl)ether	lung	inhalation
Cadmium Compounds	prostate, lung	inhalation, oral
Carbon Tetrachloride	liver	inhalation, skin
Chromium Compounds	lung	inhalation
Coke Oven Emissions	lung, urinary tract	inhalation
3,3'Dichlorobenzidine	liver, bladder	skin
Dimethyl Sulfate	respiratory	inhalation, skin
Hematite	lung	inhalation
Isopropyl Alcohol	paranasal sinuses	inhalation
4,4'-Methlylene Bis(2-Chloroaniline)	bladder	skin, inhalation
2-Naphthylamine	bladder	inhalation, oral
Nickel	nasal cavity, lung	inhalation
Polychlorinated Biphenyls	skin	skin
Soots, Tars & Mineral Oils	lung, skin, bladder	inhalation, skin
Thorium Dioxide	liver	inhalation
Vinyl Chloride	liver, brain, lung	inhalation, skin

To complicate the picture, we are constantly exposed to carcinogenic agents whether in the workplace, in the air we breathe, the food we eat and the water we drink. Since these carcinogenic agents which are present in the environment and those in the workplace may attack (even though they are different chemicals) the same cells, it is possible that many persons may have received doses much greater than the threshold presumed for any single carcinogen. This is especially true for heavy cigarette smokers. As a result, even a small exposure at the workplace could result in an increased risk of cancer.

The National Institute for Occupational Safety and Health has maintained that "exposure to any known or suspected carcinogen must be reduced to the lowest level possible by whatever means available." There is one agreement among scientists: *cancer cannot be caused if exposure to carcinogenic agents does not occur.* Although exposure to carcinogenic agents cannot be totally eliminated, the situation is far from hopeless. Not everyone will get cancer. Among those who do, many forms of cancer are curable. In addition, the risk of exposure leading to the development of cancer can be greatly reduced if proper precautions are taken such as not smoking. For fire fighters, this also means utilizing self-contained breathing apparatus (SCBA) and personal protective clothing during any emergency situation where exposure to a carcinogen may be possible.

FIRE FIGHTING AND CANCER

Epidemiological studies of fire fighters which indicate a direct correlation between exposure to a carcinogenic agent and the onset of cancer are rare. Unlike many other occupations, fire fighters are constantly entering uncontrolled environments. In many instances, fire fighters are not aware of the potential toxic and carcinogenic substances that they may be exposed to.

For example, in April 1980, a fire broke out at a chemical dump site in Elizabeth, NJ. The state, which had taken over the site two years earlier, had inventoried and removed 10,000 of the approximately 50,000 drums of chemical waste present at the site. While the fire was in progress, no one was aware of what those remaining 40,000 drums contained. Fire fighters fought the fire for almost two days; mostly without respiratory equipment because none was available. The IAFF immediately requested NIOSH to conduct a Health Hazard Evaluation and they responded by being at the scene the next day. NIOSH found that a high percentage of fire fighters were experiencing some symptoms at the time of their medical screening, 7 to 10 days after the fire. Nose and throat irritation were the most common symptoms along with acute respiratory problems such as coughing, wheezing and shortness of breath. There was also a high prevalence of skin dematitis due to chemical contacts. However, the full health effects of this fire on the exposed fire fighters are still unknown.

Without a controlled environment, it is extremely difficult to perform an epidemiological study to determine the effect of exposure to carcinogenic agents by fire fighters. In addition, there are two other considerations that must be addressed: the "healthy worker effect" and the "dead worker effect."

The "healthy worker effect" is simply that the healthiest worker are those that are employed. The physical demands of fire fighting means that only those that can frequently meet stringent employment standards in the first place are hired. Thus, the initial population is not indicative of the population at large and can significantly alter your findings in studies which seek to determine the incidence of cancer among a specific population.

Previous studies, such as Abrams' dissertation on *Occupational Mortality Among Professional Firefighters* (1974), have shown that fire fighters live approximately 10 years less than the population in general. Since cancers can take up to 40 years to develop, in many cases the fire fighter may have died from other causes such as line-of-duty heart disease before being diagnosed as having cancer. This "dead worker effect" could result in a finding that the incidence of cancer does not increase for fire fighters even though the exact opposite may be true.

The proliferation of synthetic substances into the marketplace has added a new dimension to fire fighting. Fire fighters are increasingly exposed to known and suspected carcinogenic agents whether at a residential, hardware store, drug store, dry cleaning establishment, pesticide warehouse or chemical manufacturing plant fire. The more than 30,000 hazardous waste sites and the transportation of such hazardous substances poses still more new and significant potential health risks for fire fighters.

Fire fighters, unlike other workers, are often exposed simultaneously to multiple known or suspected carcinogens. This presents another difficulty because there is little experimental data on the synergistic effects of carcinogens. The fact that smoking greatly increases the risk of lung cancer does indicate that multiple exposures to carcinogens may indeed have such synergistic effects.

Although the length and level of exposure for fire fighters may differ from the epidemiologic studies that have been performed for workers in controlled settings, it is still apparent that fire fighters are exposed to the same type of substances that have been known to cause cancer in asbestos, textile, steel, rubber industry and other workers.

Practically every emergency situation encountered by a fire fighter has the potential for exposure to carcinogenic agents. However, fire fighters can also be exposed to carcinogenic agents when the protective clothing they wear is exposed to high heat or burns. Fire fighters can even be exposed to carcinogens through the fire extinguishing agents they utilize.

Asbestos is still commonly used as a flame resistant fabric, especially in proximity fire fighting clothing and fire blankets. Manufacturer advertisement of the availability of clothing and blankets made out of asbestos fabric is another indication of its acceptance within the fire service. Asbestos fibers can separate due to flexing and abrasion and be inhaled as a carcinogenic agent by the fire fighter.

MOCA (4,4-methylene bis(2-chloroaniline)) is primarily used in the production of solid elastomeric parts. Thus, insulation in fire fighter boots and helmets and personal flotation devices can contain MOCA. MOCA can also be found at fires involving polyurethane foams found in furniture cushions, mattresses, automobile seats and safety padded dashboards, home appliance components, jet engine turbine blades and radar systems. MOCA has been shown to be associated with liver and lung cancer in rats.

Another carcinogenic agent, carbon tetrachloride, was once used in fire extinguishers and was recommended and widely used for electrical fires. There have been several reports showing liver cancer in humans as being associated with exposure to carbon tetrachloride.

Carbon tetrachloride is still utilized as a metal degreaser, refrigerant and grain fumigant.

The list of potential carcinogenic agents that fire fighters can be exposed to is almost as long as the list of all known or suspected carcinogens. Among the more common substances to which fire fighters are potentially exposed include asbestos, creosote, polychlorinated biphenyls, plastics and pesticides. Another new danger is the cancer hazard caused by radiation exposure.

Asbestos

Asbestos is a mineral that appears in a fibrous and fluffy form when separated from rock into fibers of differing length. These fibers are resistant to heat, acid, corrosion and possess the ability to absorb and filter.

Asbestos has been widely used in many industries as insulation and fireproofing. Currently, there are more than 3,000 products, mostly in the construction industry, that are made using asbestos. There are four major types of asbestos that are commonly encountered by fire fighters: amosite, anthophyllite, chrysotile and crocidolite.

TYPES AND USES OF ASBESTOS

Asbestos Fiber	*Fiber Color*	*Common Uses*
amosite	brown, grey, green or yellow	cement pipe, cement sheet, roofing products, thermal insulation.
anthophyllite	brown, grey, green or yellow	cement pipe, packing and gaskets, plastics, paper
chrysotile	white, grey, green or yellowish	cement pipe, cement sheet, flooring products, roofing products, packing and gaskets, thermal insulation, electrical insulation, paper friction products, coatings and compounds, plastics, textiles
crocidolite	blue	cement pipe, packing and gaskets, plastics, paper

Fire can destroy the surrounding material in which asbestos fibers are embedded, thus releasing them into the environment. The fibers themselves do not burn. During overhaul operations, fire fighters tear apart burned structures searching for fire extension. This process releases asbestos fibers from the torn insulation and construction materials into the air. Fans used to vent the fire scene during overhaul may cause these fibers to spread even more.

Asbestos is dangerous when the fibers are released into the air and inhaled or swallowed. Asbestos fibers are microscopically small; each fiber is hundreds of times smaller than a human hair. The fibers are so fine that they can float in the air indefinitely without settling. These small fibers can easily enter the lungs where they can remain for life. There are two types of diseases that can result from breathing or swallowing asbestos fibers: asbestosis and several forms of cancer.

Asbestos exposure is known to increase occurrence of lung and wind pipe cancer, cancer of the large intestine and is chiefly linked to occurrences of mesothelioma (a rare cancer of the chest and abdominal lining). It is also known that smoking considerably increases the risk of persons who work around asbestos. Despite the high occurrences, not everyone exposed to asbestos will get cancer.

Asbestos exposure will not produce any immediate adverse health effects. Such adverse health symptoms may not occur for 20, 30 or 40 years after exposure. Symptoms of asbestos-related diseases, once they become apparent, include shortness of breath, coughing, blood in the fluid coughed up from your lungs, pain in the chest or abdomen, difficulty in swallowing and rapid large weight loss.

Although the longer the exposure to asbestos the more apt one is to get cancer, studies have shown that a two- or three-month exposure can cause the onset of mesothelioma. Fire fighters who have worked for long periods without utilizing SCBA, such as during overhaul, could have had a similar exposure experience as those short-term asbestos workers.

Creosote

Creosote (creosotum, creosote oil, brick oil) is a complex mixture of organic chemicals that appears in the form of a thick, tarry liquid or semi-solid substance. The main uses of creosote are as a wood preservative and in pitch for roofing. Creosote itself is a carcinogenic agent found in soot, tars and mineral oils. The 1775 study by Pott of chimney sweeps in England who developed scrotal cancer was due to a creosote-like substance.

Creosote has been used as an antiseptic, disinfectant, germicide, constituent of fuel oil and a therapeutic agent. Fire fighters may encounter creosote on the waterfront, from burning wharves, dock pilings and other wood on or near the water. Other common sources include utility poles, and/or other woods treated with creosote to prevent decay, mildew or other type of corrosion. Creosote in wood can be recognized through its tarry smell and dark or even black color.

Creosote, which burns at 122° F (50° C), also gives off a carcinogen called benzo(a)pyrene and many other related compounds known as polycyclic aromatic hydrocarbons (PAH). Exposure from creosote can occur by inhaling fumes as it burns and/or by skin absorption. Creosote is associated with cancer of the skin, forearms, prostate, testicles and penis. Exposure to benzo(a)pyrene has been associated with cancers of the mouth, throat, windpipe and lung.

After exposure to creosote, the skin may become reddish, burn, itch, turn a grayish or bronze color in areas, blister, ulcerate or even turn gangrenous. The fire fighter's eyes may be injured, producing an inflammation of the mucous membrane lining the inner surface of the eye or permanently scarring the cornea. Other acute health effects include salivation, vomiting, dizziness, headache, hypothermia, a bluish discoloration of the skin due to the lack of sufficient oxygen in the blood, convulsions, weak pulse, breathing difficulties and a skin rash.

There are no special tests to detect exposure to creosote. However, if there is a rash or other abnormalities on the skin, then a physician should be consulted to determine whether a biopsy is required.

Polychlorinated biphenyls

Polychlorinated biphenyls (PCBs) are a group of heavy, oily, liquid organic chemicals. PCBs are synthetic chemicals produced during a chemical reaction using chlorine and certain petroleum derivatives. There are more than 200 members of this chemical group known as PCBs, but the most common forms are chlorodiphenyl made up of either 42% chlorine or 54% chlorine. PCBs range in appearance from a straw-colored, oily liquid to a white or yellowish waxy solid depending on the amount chlorinated. PCBs from a capacitor or transformer that has exploded may be black in color. PCBs are flame resistant, but they do begin to give off vapors at 122° F (50° C). At high temperatures such as encountered in a fire, liquid PCBs give off toxic vapors.

PCBs, which are chemically inert, nonflammable, resistant to heat and pressure, and electrically nonconducting, are extremely attractive for industrial uses. PCBs are found wherever there are transformers or capacitors. These can range from electrical transformers in buildings and at utility company facilities to capacitors in television sets, fluorescent lights and home air conditioners. Any transformer or capacitor containing an oily liquid or a white or yellowish solid is likely to contain PCBs. PCBs are also used as an additive for extreme pressure lubricants (e.g., hydraulic systems, vacuum pumps and gas transmission turbines), as a coating for investment casting molds in foundries and in carbonless copying papers.

Although PCB production was restricted in 1971 and banned by the Environmental Protection Agency in 1977, equipment using PCBs is still in widespread use. Fire fighters should assume that any capacitor and any fluid-filled transformer contains PCBs or PCB-contaminated fluid. At high temperatures, PCBs also form other extremely hazardous substances such as dioxins (used in Agent Orange) and polychlorinated dibenzofurans.

PCBs have been marketed commercially since 1929 under trade names such as Abestol, Acroclor, Chlorextol, Clorhen, Kanechlor, Inerteen, No-Flamol, Phenoclor, Pyranol and the familiar Askarel. Fire fighters encountering PCBs in a transformer will usually see it labeled as Askarel or Acroclor.

PCBs enter the body through inhalation of air contaminated with vapors, mists or particulates containing PCBs. They can also enter through the skin or eye contact with materials containing PCBs and/or by swallowing food or other materials contaminated with PCBs. For example, PCBs can readily penetrate the neoprene vapor barrier commonly used in fire fighter protective clothing. Once absorbed into the body, they tend to settle in the liver and fat cells.

PCBs are suspected to be associated with liver and pancreas cancer in humans. PCB exposure has also been associated with decreased sperm count, impotence and other reproductive problems; damage to the nervous system causing tremors; and liver damage.

Exposure to PCBs may produce irritation to the eyes, nose and throat as well as water retention and swelling, jaundice (if liver damage has occurred), vomiting, weight loss, loss of appetite, abdominal pains and fatigue. Exposure to PCB fumes may cause the onset of chloracne, a severe and painful skin rash.

Plastics

Plastics are long chains of organic molecules made through a linkage process known as polymerization. During the last two decades, the rapid proliferation of plastic products entering the marketplace has added a new dimension to fire fighting. The presence of plastics can probably be expected at every fire emergency, because of the variety of products that are made such as furniture, electric wire insulation, office equipment and kitchen gadgets. It is estimated that there are over 30 billion pounds of plastic made annually in the United States alone. Of these plastics, about 13 billion pounds is polyethylene and 6 billion pounds is polyvinylchloride.

For a fire fighter, the problems with plastics begin when they are heated. As plastic heats up, it begins to break down into different chemical elements. These elements which are given off as fumes may be odorless and colorless and are sometimes toxic and carcinogenic. Toxic fumes begin to be given off long before the plastic actually

catches fire. These fumes, as dramatized in the MGM Grand Hotel fire, when inhaled can cause death far from the actual fire site.

Polyvinylchloride (PVC) is a mixture of vinyl chloride and a variety of other additives. The particular mixture will depend upon the manufacturer and the intended purpose. Likewise, the fumes that are given off will depend upon the type of PVC and the temperature. As PVC heats up, vinyl chloride may be released. When PVC burns, benzene, hydrogen chloride, phosgene, carbon monoxide and carbon dioxide are also given off.

PRODUCTS OF SEVERAL COMMON PLASTICS

Plastic	*Typical Products*
ABS	piping, luggage, automobile dashboards, calculator housings, refrigeration liners, margarine tubs
Phenolics	circuit breakers, distributor caps, automobile steering wheels, fuse blocks, pot handles
Polycarbonate	helmets (football/fire fighter/baseball), power tool housings, battery cases, safety glasses, molded products
Polyethylene	milk bottles, seats, waste baskets, disposal syringes, pallets, shipping pails, trash bags, packaging lids, communication cables, bowls, garment bags, wire/cable coatings
Polypropylene	auto fender skirts, battery cases, carpet packing, dishwasher tubs, door liners, radio/tv/phonograph housings
Polystyrene	foam and nonfoam cups, interior doors, margarine tubs, appliances, shutters
Polysulfonate	coffee makers, camera bodies, electrical connectors, battery cases
Polyurethane	cushioning for furniture, mattresses and bed pillows, carpet pads, building insulation, refrigerator and freezer insulation, structural portions of chairs, tables, cabinets, picture frames, decorative beams and wall panels, swimming pools, sporting goods
yvinylchloride	phonographic records, bottles, piping, siding, wall covering, flooring, upholstery, chemical wire coating

The raw material of PVC, vinyl chloride is one of the top fifty chemicals produced in the United States. Vinyl chloride and benzene are known or suspected carcinogenic agents. Vinyl chloride has been associated with cancers of the liver, brain, lung, blood and nervous system. Benzene has been associated with increasing the risk of leukemia. In addition to being a combustion by-product of PVC, polystyrene, polyurethane and other plastics, benzene is used as a constituent in motor fuels, as a solvent for fats, inks, oils, paint, plastics and rubber, in photogravure printing and as a chemical intermediate.

The degradation of the plastic polyurethane produces hydrogen cyanide gas and urethane, a probable human carcinogen. Polyurethane also produces acrylonitrile, which has been associated with increased incidences of respiratory and colon cancers. Acrylonitrile is also used in the manufacturing of synthetic fibers, acrylonitrile-butadiene-styrene (ABS) plastics, nitrile rubbers, chemicals and adhesives. In addition, acrylonitrile has been used as a pesticide.

ACUTE SYMPTOMS OF PLASTIC EXPOSURE

Carcinogen	*Immediate Symptoms*
acrylonitrile	Irritation of the eyes. Repeated and lengthy exposure may produce skin irritation. Blistering may occur after prolonged contact with the skin. May also produce nausea, vomiting, headaches, sneezing, and light-headedness and weakness.
benzene	Irritation to the skin, eyes and upper respiratory tract. May result in blistering in or beneath the skin, redness of the skin, and a dry, scaly, cracked rash. Exposure to extremely high concentrations results in central nervous system depression, headache, dizziness, nausea, convulsions and coma may occur. Continuing exposure causes changes in blood and in bone marrow.
urethane	Irritation of the eyes, respiratory tract and skin; may be severe enough to produce bronchitis and fluid in the lungs. May create an asthmatic reaction. Exposure over a long period of time may produce a decrease in breathing capacity.
vinyl chloride	Symptoms resemble mild alcohol intoxication. Lightheadness, some nausea, vomiting and dulling of seeing and hearing responses may develop with a very high level of exposure. Liver damage may occur, also eye damage.

Attachment #1-24

Probably the most dangerous period of exposure to the by-products of plastic combustion for the fire fighter is during overhaul. This occurs because fire fighters frequently remove their respiratory protection leaving them exposed to the fumes that may linger for hours, especially in confined spaces.

Pesticides

Pesticides are found everywhere, in grocery stores, residential homes, drug stores, hardware stores, garden and flower shops, as well as agricultural sites. Pesticides known or suspected of being carcinogenic include chlordane, heptachlor, dieldrin, DDT, kepone, lindane, mirex, toxaphene and so forth.

Chlordane was used as an insecticide on preplanting soil, fire ants and harvester ants prior to being banned in the mid-1970's.

Heptachlor is used as an insecticide in seed treatment, preplanting soil application, dipping tops of plants and roots for control of insects, flies and mosquitoes. It is also used on household plants and on agricultural crops and fruits.

Kepone was first introduced in 1958 and has been used as an insecticide against leaf-eating insects, ants, cockroaches and as a larvicide against flies. In the late 1970's the production and use of kepone was stopped in the United States. Research studies have shown an increased incidence of heptatocellular cancers in rats and mice.

Lindane is the accepted common name for a group of gamma isomers of hexachlorocyclohexane. Lindane is primarily used for insecticidal treatment of hardwood logs and lumber, seed grains and livestock. Secondary uses of lindane include its application as an insecticide on several dozen fruits and vegetable crops. Exposure to lindane in humans have shown increased incidence of leukemia and lung tumors.

Mirex has been used extensively to control the fire ant, especially in the southeastern region of the United States. Mirex has also been used to treat other species of ants and yellow jackets. The use of mirex as a pesticide was discontinued in the late 1970's. In animal studies, mirex has caused an excess of liver tumors.

Toxaphene is one of the most popularly used pesticides. The primary use of toxaphene is to control cotton insect pests. It is also used to control insect pests on livestock, poultry and a few field crops (soybeans, peanuts). In the United States, the southeast and delta states are responsible for most of the toxaphene used. In animal studies, toxaphene has been shown to produce liver cancers in [illegible].

Pesticides can affect the body if inhaled, if they come in contact with the eyes or skin or if they are swallowed. Like PCBs, pesticides

may penetrate neoprene vapor barriers commonly utilized in fire fighter protective clothing. Mild poisoning after exposure can cause symptoms such as dizziness, nausea, abdominal pain and vomiting. Moderate poisoning can show the same symptoms as mild poisoning followed by severe irritability, convulsive seizures and coma. In severe cases, the convulsions may be continuous with rapid heart beat, labored breathing, unconsciousness and eventually death.

Radiation

Although we have always been exposed to minute amounts of radiation during our daily lives, exposure to high levels of radiation is a relatively new danger brought about by the use of atomic energy for peaceful uses.

While radiation is a form of energy rather than a chemical or metal, particles of radioactive substances can be found in dust or smoke. Radiation is emitted, transmitted or absorbed in a wave or energetic particle form. The most hazardous form of radiation is ionizing radiation which severely damages the body's cells and tissues.

Ionizing radiation is produced naturally through the decay of radioactive elements or artificially through X-ray machines and other devices. Fire fighters can encounter ionizing radiation when responding to emergencies at factories that produce drugs, fire alarms, X-ray tubes, electronic tubes or in medical offices, hospitals, television repair shops, petroleum refineries and scientific research laboratories.

Radiation is unique because of its ability to directly enter the body through the skin much like sunlight going through a window. This direct route of exposure is an external hazard. Internal hazards are caused by radioactive materials entering our bodies through inhalation, ingestion and skin absorption. Generally, radioactive materials enter the body under occupational conditions primarily through inhalation. However, a skin puncture or laceration could result in radioactive particles being implanted under the skin. In addition, contamination of a fire fighter's turnout, helmet, boot or gloves with radioactive materials can result in accidental ingestion of radioactive particles or dust. Experience has shown that workers exposed to radiation have high rates of occupational illnesses such as cancer, leukemia, sterility, cataracts and life span shortening. In addition, such exposure could also have teratogenic and mutagenic effects. Thus, infants born of mothers after exposure to the atomic bomb had an increased incidence of malformation and abnormality of the central nervous system.

The most common forms of ionizing radiation encountered are alpha, beta, gamma and X-rays. Alpha radiation cannot penetrate the skin, thus is not an external hazard. However, alpha-emitting materials can be inhaled into the body with serious consequences. Beta

radiation can travel into the tissues of the body, however, it usually cannot penetrate through a fire fighter's protective clothing. Exposure through inhalation is again the most severe hazard. Both gamma rays and X-rays are primarily external hazards, that is, they readily penetrate the skin surface. Gamma emitters can also pose serious hazards through inhalation or ingestion.

Radiation exposure represents one of the most severe cancer causing hazards. Like chemical carcinogens, radiation exposure is dose dependent with some risks even at the lowest measurable exposure level. Again, in a manner similar to exposure to chemical carcinogens, exposure to radiation may not produce any immediate adverse health effects. Radiation exposure can also be followed by a latency period that can last several decades.

RADIATION LATENCY PERIOD

Cancer Effect	*Time Elapsed From Initial Radiation*
Leukemia	5-30 years
Bone Cancer	5-30 years
Lung Cancer	10-50 years
Other Cancers	Variable years

GUIDELINES FOR DETERMINING OCCUPATIONALLY-RELATED CANCER

With the fight for presumptive cancer legislation just beginning, case by case determinations must be made for successful workers' compensation, disability or pension cases. In contrast to a traumatic injury that is readily apparent to everyone, the relationship between the profession of fire fighting and cancer may not always be clear cut. Cancers are usually slow to develop and may not appear until after an individual retires. In addition, it is frequently difficult to document exposures of an individual to carcinogenic agents.

Cancers develop very slowly in humans, usually manifesting themselves from 5 to 40 years after exposure to the cancer-causing agent. For example, cancers of the liver, lung or bladder may not appear until 30 years after exposure to asbestos, vinyl chloride or benzidine. This long period of latency is one of the major reasons why it is so difficult to determine the exact causes of cancers. It is also difficult to document occupational exposure to carcinogens because of the inability to pinpoint the specific agent or agents and the level of exposure that a worker may be exposed to.

Decisions in workers' compensation and other similar cases are generally based on an evaluation of the available information. When evidence is presented in an organized and logical fashion, when major issues are clearly identified and the causal factors are indicated, then the greater the likelihood of a favorable and equitable decision.

In such documented cases, the relationship between cancer and fire fighting has been constantly acknowledged. The most prominent example is the State of California's enactment of presumptive cancer legislation (Appendix II). This law presumes that cancer is occupationally related when it can shown that exposure to a carcinogen during employment took place which can be reasonably linked to the cancer.

Workers' compensation boards have already identified cancer in fire fighters which is employment-related. An occupational medical specialist, John B. Webster, M.D., found that an Ohio fire fighter's leukemia was caused by his exposures to carcinogens. In San Francisco, the Retirement Board ruled that a fire fighter's death from intestinal cancer was occupationally related. Other studies have shown excessive incidences of buccal, pharynageal, intestinal, rectal and colon cancer in fire fighters. A study in Toronto over the course of 25 years found that cancer increased steadily as a cause of death, from 15.4% in 1945-9 to 38.4% in 1967-70, among active fire fighters. A NIOSH study of mortality in Washington State over a twenty-year period found that fire fighters had a higher incidence of lymphatic leukemia and cancers of the lympatic and hematopoietic

sues. Thus, the epidemiological work performed so far does suggest that cancer is an occupational disease afflicting fire fighters.

For jobs such as fire fighting, any stress may be an aggravating factor. Since most states hold that the employer accepts the worker "as is" such factors as age, sex, heredity and obesity can be excluded from the list of causative factors. This basically leaves those mechanical, chemical, physical or biological exposures in the working and nonworking environment to be considered. Although this is an easy and simple summation, aggravation cases frequently have multiple causes, many of which are either unknown or not understood.

General speaking, a cancer is judged to be occupationally related if the following conditions are apparent.

1. The worker's occupational environment (past or present) involved exposure to an agent or agents sufficient to have caused the onset of the disease in question.
2. The worker's disease is compatible to the type of agent or agents they were exposed to.
3. The weight of the evidence supports that the onset of disease was occupationally-related.

To answer these questions, NIOSH has developed a criteria of information that needs to be compiled and assembled.

Medical History

- onset of present illness
- all previous illnesses (childhood, physical, mental)
- injuries
- surgical procedures
- hospital admissions

Personal history

- age, sex, marital status, number of children
- name and location of all places of residence since birth
- locations visited prior to onset of symptoms
- alcohol and tobacco use
- medications or drug use
- recreation and hobbies
- use of chemicals in the home (cleaning agents, aerosols, etc.)
- details specific to any suspected occupational causative agent(s)

Family History

- age, sex and health status of worker's parents, siblings, spouse and children
- any chronic or occupational disease in the family or household

Occupational History

- past and present job titles
- complete listing of actual work performed
- duration of each type of activity
- dates of employment and worker's age for each job activity
- geographic and physical location of employment
- type of personal protective clothing and equipment used and frequency
- type of agents or substances to which worker has been exposed, include frequency and average duration of each exposure situation

Clinical Evaluation

- routine examination of all physiological systems
- evaluation of behavior as related to emotional status
- specific examination for health effects of suspected disease agents
- comparison of date of onset of symptoms with occupational history
- evaluation of results of any past biological or medical monitoring (blood, urine or any other sample analysis) and previous physical examinations
- evaluation of laboratory tests: routine (complete blood count, blood chemistry profile, urinalysis) and specific for suspected disease agents (blood or urine test for specific agent, chest or other X-rays, liver function tests, pulmonary function tests)

Evidence of Exposure

- expert testimony (industrial hygienist) should be obtained concerning general environmental conditions, especially if air sampling data is not available
- establishment of the precise disease-causing agents that the worker was exposed to

- a complete description of the worker's duties, including type of protective equipment utilized and type of protective clothing worn
- information concerning the modes of entry of the agent into the body (inhalation, skin absorption)

Once the information and appropriate documentation has been obtained and evaluated, then the following questions represent a guideline to determine the equity of the claim:

1. Has a disease condition been clearly established?
2. Has it been shown that the disease can result from the suspected agent(s)?
3. Has exposure to the agent(s) been demonstrated by work history, expert opinion or sampling data?
4. Has exposure to the agent been shown to be of sufficient degree and/or duration to result in the disease condition by scientific literature, epidemiologic studies?
5. Has nonoccupational exposure to the agent been ruled out as a causative factor?
6. Have all special circumstances been weighed?

If the answer to all of these questions is "yes," then the burden of proof has been met and it can generally be concluded that the disease is occupational in origin.

CONCLUSION: PREVENTION

Fire fighters know that their profession is the most hazardous. It is also time to recognize that fire fighting is different today than it was twenty years ago. The environment a fire fighter must encounter has changed. Fire fighters must frequently respond to emergency situations without adequate knowledge of either the type of physical, chemical or biological agents they may encounter or their toxicity.

Recent death and occupational disease statistics compiled by the International Association of Fire Fighters clearly substantiates that there is a new dimension to fire fighting. Reported deaths and forced retirements due to occupational related diseases are growing at a dramatic rate. This survey has reported that cancer deaths make up more than one-third of all those reported due to occupational illnesses.

The "smoke-eater" simply cannot survive in the modern fire environment. Almost every emergency situation to which a fire fighter must respond requires the use of a SCBA. It has been well-documented that "positive-pressure" or "pressure-demand" SCBA offers the best type of protection for the fire fighter. However, a SCBA can only protect if it is properly utilized and maintained.

Fire fighters also must try to obtain more knowledge about the environments they must confront. For fire fighters, there is no federal standard that can effectively regulate the amount of exposure they may have to a carcinogen, since the environment they enter is uncontrolled. Nevertheless, fire fighters are required on a daily basis to constantly work under such conditions, many times without sufficient quantities of "positive-pressure" SCBA or adequate personal protective clothing. Fire fighters have the "right to know" through proper labeling and pre-fire planning the type of carcinogens they can potentially be exposed to when they are asked to respond to an emergency situation.

Protection and knowledge are necessary ingredients to prevent the occurrence of occupational illnesses, such as cancer. However, the nature of the profession of fire fighting can never completely eliminate exposure to carcinogenic agents or the need to perform strenuous activities under the worse conditions imaginable. Medical research has progressed to the point that cancer does not automatically mean death. In fact, almost half of all cancer patients can be cured with currently available treatments. In many cases, early detection is the key. This means that routine medical examinations for fire fighters could be vital in preventing unnecessary premature deaths.

APPENDIX I

LIST OF KNOWN OR SUSPECTED CARCINOGENS

SUBSTANCE	SYNONYMS
2-Acetylamino-fluorene	AAF * 2AAF * ACETAMIDE, N-9H-FLUOREN-2-YL-(9Cl) * 2-ACETAMINOFLUORENE * N-ACETYL-2-AMINOFLUORENE * 2-(ACETYLAMINO)FLUORENE 2-ACETYLAMINOFLUORENE * FAA * 2-FAA * 2-FLUORENYLACETAMIDE * N-2-FLUORENYLACETAMIDE * N-FLUOREN-2-YLACETAMIDE
Acrylonitrile	ACRYLON * ACRYLONITRILE (DOT) * ACRYLONITRILE MONOMER * CARBACRYL * CYANOETHYLENE * ENT 54 * FUMIGRAIN * MILLER'S FUMIGRAIN * PROPENENITRILE * 2-PROPENENITRILE * TL 314 * VCN * VENTOX * VINYL CYANIDE
Aflatoxins	NONE
Aldrin	ALDREX * ALDRIN * ALDRIN (DOT) * ALDRITE * ALDROSOL * COMPOUND 118 * DRINOX * ENT 15,949 * HEXACHLOROHEXAHYDRO-endo-exo-DIMETHANONAPHTHALENE * 1,2,3,4,10,10-HEXACHLORO-1,4,4a,5,8,8a-HEXAHYDRO-1,4,5,8-DIMETHANONAPHTHALENE * 1,2,3,4,10,10-HEXACHLORO-1,4,4a,5,8,8a-HEXAHYDRO-exo-1,4-endo-5,8-DIMETHANONAPHTHALENE * 1,2,3,4,10,10-HEXACHLORO-1,4,4a,5,8,8a-HEXAHYDRO-1,4-endo-exo-5,8-DIMETHANONAPHTHALENE * HHDN * NCI-C00044 * OCTALENE * SEEDRIN
4-Aminobiphenyl	p-AMINOBIPHENYL * p-AMINODIPHENYL * 4-AMINODIPHENYL * BIPHENYLAMINE * [1,1'-BIPHENYL)-4-AMINE * p-BIPHENYLAMINE * PARAAMINODIPHENYL * p-PHENYLANILINE * XENYLAMINE
Amitrole	AMEROL * AMINOTRIAZOLE * 3-AMINOTRIAZOLE * 3-AMINO-s-TRIAZOLE * 3-AMINO-1,2,4-TRIAZOLE * 2-AMINO-1,3,4-TRIAZOLE * 3-AMINO-1H-1,2,4-TRIAZOLE * AMINOTRIAZOLE (PLANT REGULATOR) * AMINO TRIAZOLE WEEDKILLER 90 * AMINOTRIAZOL SPRITZ-PULVER * AMITOL * AMITRIL * AMITRIL T L * AMITROL * AMITROL 90 * AMITROL-T * AMIZOL * AMIZOL D * AMIZOL DP NAU * AMIZOL F * AT * 3,A-T * ATA * AZAPLANT * AZAPLANT KOMBI * AZOLAN * AZOLE * CAPAPRIM A 1544 * CYTROL * CYTROL AMITROLE-T * CYTROLE * DIUROL * DIUROL 5030 * DOMATOL * MOMATOL 88 * ELMASIL * EMISOL * EMISOL 50 * EMISOL F * ENT 25445 * FENAMINE * FENAVAR * HERBIDAL TOTAL * HERBIZOLE * KLEER-LOT * ORGA-414 * RADOXONE TL * RAMIZOL * SIMAZOL * SOLUTION CONCENTREE T271 * TRIAZOLAMINE * 1H-1,2,4-TRIAZOL-3-AMINE * USAF XR-22 * VOROX * VOROX AA * VOROX AS * WEEDAR ADS * WEEDAR AT * WEEDAZIN * WEEDAZIN ARGINIT * WEEDAZOL * WEEDAZOL GP2 * WEEDAZOL SUPER * WEEDAZOL T * WEEDAZOL TL * WEEDEX GRANULAT * WEEDOCLOR * X-ALL LIQUID

SUBSTANCE	SYNONYMS
Aramite	ACARACIDE • ARACIDE • ARAMITE-15W • ARATRON • BUTYLPHENOXYISOPROPYL CHLOROETHYL SULFITE • 2-(p-BUTYLPHENOXY)ISOPROPYL 2-CHLOROETHYL SULFITE • 2-(4-1-BUTYLPHENOXY)ISOPROPYL-2-CHLOROETHYL SULFITE • 2-(p-t-BUTYLPHENOXY)ISOPROPYL 2'-CHLOROETHYL SULPHITE • 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL 2-CHLOROETHYL ESTER of SULPHUROUS ACID • 2-(p-BUTYLPHENOXY)-1-METHYLETHYL 2-CHLOROETHYL SULFITE • 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL 2'-CHLOROETHYL SULPHITE • 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL SULPHITE of 2-CHLOROETHANOL • CES • beta-CHLOROETHYL-beta-(p-t-BUTYLPHENOXY)-alpha-METHYLETHYL SULFITE • beta-CHLOROETHYL-beta-(p-t-BUTYLPHENOXY)-alpha-METHYLETHYL SULPHITE • 2-CHLOROETHYL 1-METHYL-2-(p-t-BUTYLPHENOXY)ETHYL SULPHATE • 2-CHLOROETHYL SULPHITE of 1-(p-t-BUTYLPHENOXY)-2-PROPANOL • COMPOUND 88R • ENT 16,519 • ETHANOL, 2-CHLORO-, 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL SULFITE • ETHANOL, 2-CHLORO-, ESTER WITH 2-(p-tert-BUTYLPHENOXY)-1-METHYLETHYL SULFITE • NIAGARAMITE • ORTHO-MITE • 2-PROPANOL, 1-(p-t-BUTYLPHENOXY)-, 2-CHLOROETHYL SULFITE • 88-R
Arsenic & Certain Arsenic Compounds	ARSENIC BLACK • ARSENIC, SOLID (DOT) • COLLOIDAL ARSENIC • GREY ARSENIC • METALLIC ARSENIC
Asbestos	ACTINOLITE • AMIANTHUS • AMOSITE • AMPHIBOLE • ANTHOPHYLLITE • ASBESTOS FIBER • ASBESTOS FIBRE • ASCARITE • CHRYSOTILE • CROCIDOLITE • TREMOLITE
Auramine	APYONINE AURAMINE BASE • AURAMINE BASE • AURAMINE (FREE BASE) • AURAMINE N BASE • AURAMINE OAF • AURAMINE O BASE • AURAMINE OO • AURAMINE SS • AUREMINE • BENZENAMINE,4,4'-CARBONIMIDOYLBIS(N,N-DIMETHYL-(9Cl) • BIS,p-DIMETHYLAMINOPHENYL)METHYLENEIMINE • BRILLIANT OIL YELLOW • C.I. 41000B • C.I. BASIC YELLOW 2, FREE BASE • C.I. SOLVENT YELLOW 34 • 4,4'-DIMETHYLAMINOBENZOPHENONIMIDE • GLAURAMINE • 4,4'-(IMIDOCARBONYL)BIS(N,N-DIMETHYLANILINE) • TETRAMETHYLDIAMINODIPHENYLACETIMINE • WAXOLINE YELLOW O • YELLOW PYOCTANINE
Azaserine	ACETIC ACID, DIAZO-, ESTER WITH SERINE • L-AZASERINE • DIAZOACETATE (ESTER) L-SERINE • L-DIAZOACETATE (ESTER) SERINE • O-DIAZOACETYL-L-SERINE • NSC-742
Benz(a)anthracene	BA • 1,2-BENZANTHRACENE • BENZANTHRENE BENZO(a)ANTHRACENE • BENZO(a)PHENANTHRENE • BENZO(b)PHENANTHRENE • 2,3-BENZOPHENANTHRENE • 2,3-BENZPHENANTHRENE • NAPHTHANTHRACENE • TETRAPHENE

SUBSTANCE	SYNONYMS
Benzene	(6)ANNULENE • BENZENE (DOT) • BENZIN • BENZINE • BENZOL • BENZOLE • BENZOLENE • BICARBURET of HYDROGEN • CARBON OIL • COAL NAPTHA • CYCLOHEXATRIENE • MINERAL NAPTHA • MOTOR BENZOL • NCI-C55276 • NITRATION BENZENE • PHENE • PHENYL HYDRIDE • PYROBENZOL • PYROBENZOLE
Benzidine	BIPHENYL, 4-4'-DIAMINO- • 4,4'-BIPHENYLDIAMINE • C.I. AZOIC DIAZO COMPONENT 112 • 4,4'-DIAMINOBIPHENYL • p-DIAMINODIPHENYL • 4,4'-DIAMINODIPHENYL • p,p'-DIANILINE • 4,4'-DIPHENYLENEDIAMINE • FAST CORINTH BASE B
Benzo(b)fluoranthene	FLUORANTHENE • 3,4-BENZFLUORANTHENE • 3,4-BENZ(•)ACEPHENANTHRYLENE • 2,3-BENZ-2,3-BENZOFLUORANTHENE • BENZO(•)FLUORANTHENE • 3,4-BENZOFLUORANTHENE • 2,3-BENZOFLUORANTHENE • B(b)F
Benzo(j)fluoranthene	BENZ(i)FLUOROANTHRENE • 10,11-BENZFLUORANTHENE • 7,8-BENZOFLUORANTHENE • 10,11-BENZOFLUORANTHENE • B(i)F • DIBENZO(a,jk)FLUORENE
Benzo(a)pyrene	3,4-BENZOPYRENE • 6,7-BENZOPYRENE • 3,4-BENZPYRENE • 3,4-BENZ(a)PYRENE • 3,4-BENZYPYRENE • BP • B(a)P • 3,4-BP
Beryllium & Certain Beryllium Compounds	BERYLLIUM-9 • GLUCINIUM • GLUCINUM
N,N-Bis(2-chloroethyl)-2-naphthylamine	2-BIS(2-CHLOROETHYL)AMINONAPHTHALENE • BIS(2-CHLOROETHYL)-beta-NAPHTHYLAMINE • CHLORNAFTINA • CHLORNAPHAZIN • CHLORNAPHAZINE • CHLORNAPHTHIN • CHLORONAFTINA • CHLORONAPHTHINE • DICHLOROETHYL-beta-NAPHTHYLAMINE • DI(2-CHLOROETHYL)-beta-NAPHTHYLAMINE • NN-DI(2-CHLOROETHYL)-beta-NAPHTHYLAMINE • 2-N,N-DI(2-CHLOROETHYL)NAPHTHYLAMINE • ERYSAN • NAPHTHYLAMINE MUSTARD • beta-NAPHTHYL-BIS-(beta-CHLOROETHYL)AMINE • 2-NAPHTHYLBIS(2-CHLOROETHYL)AMINE • beta-NAPHTHYL-DI-(2-CHLOROETHYL)AMINE • NSC-62209 • R48
Bis(chloromethyl)ether	BIS-CME • BCME • CHLORO(CHLOROMETHOXY) METHANE • CHLOROMETHYL ETHER • sym-DICHLORO-DIMETHYL ETHER • sym-DICHLOROMETHYL ETHER • DIMETHYL-1,1'-DICHLOROETHER • METHANE, OXYBIS(CHLORO- • OXYBIS(CHLOROMETHANE)
Cadmium & Certain Cadmium Compounds	C.I. 77180

SUBSTANCE	SYNONYMS
Carbon Tetrachloride	BENZINOFORM * CARBONA * CARBON CHLORIDE * CARBON TET * CARBON TETRACHLORIDE (DOT) * ENT 4,705 * FASCIOLIN * FLUKOIDS * FREON 10 * HALON 104 * METHANE TETRACHLORIDE * METHANE, TETRACHLORO- * NECATORINA * NECATORINE * PERCHLOROMETHANE * TETRACHLOORMETAAN * TETRACHLOROCARBON * TETRACHLOROMETHANE * TETRAFINOL * TETRAFORM * TETRASOL * UNIVERM * VERMOESTRICID
Chlorambucil	AMBOCHLORIN * 4-(BIS(2-CHLOROETHYL)-AMINO)BENZENEBUTANOIC ACID * gamma-(p-BIS(2-CHLOROETHYL)AMINOPHENYL)-BUTYRIC ACID * 4-(p-(BIS(2-CHLOROETHYL) AMINO)PHENYL)BUTYRIC ACID * 4(p-BIS(beta-CHLOROETHYL)AMINOPHENYL)BUTYRIC ACID * CB 1348 * CHLORAMINOPHEN * CHLORAMINOPHENE * CHLOROBUTIN * CHLOROBUTINE * N,N-DI-2-CHLOROETHYL-gamma-p-AMINOPHENYLBUTYRIC ACID * p-(N,N-DI-2-CHLOROETHYL)AMINOPHENYL BUTYRIC ACID * p-N,N-DI-(beta-CHLOROETHYL)AMINOPHENYL BUTYRIC ACID * gamma-(p-DI(2-CHLOROETHYL)-AMINOPHENYL)BUTYRIC ACID * ELCORIL * LEUKERAN * LEUKERSAN * UNFOLYSIN * NCI-C03485 * NSC-3088 * PHENYLBUTYRIC ACID NITROGEN MUSTARD
Chlordane	ASPON-CHLORDANE * BELT * CD 68 * CHLORDAN * gamma-CHLORDAN * CHLORDANE,
Chlordane (Cont'd)	LIQUID (DOT) * CHLORINDAN * CHLOR KIL * CHLORODANE * CORODANE * CORTILAN-NEU * DICHOLORCHLORDENE * DOWCHLOR * ENT 9,932 * ENT 25,552-X * HCS 3260 * KYPCHLOR * M 140 * M 410 * 4,7-METHANO-1H-INDENE, 1,2,4,5,6,7,8,8-OCTACHLORO-2,3,3a,4,7,7a-HEXAHYDRO- * NCI-C00099 * NIRAN * OCTACHLOR * OCTACHLORODIHYDRODICYCLOPENTADIENE * 1,2,4,5,6,7,8,8-OCTACHLORO-2,3,3a,4,7,7a-HEXAHYDRO-4,7-METHANOINDENE * 1,2,4,5,6,7,8,8-OCTACHLORO-2,3,3a,4,7,7a-HEXAHYDRO-4,7,-METHANO-1H-INDENE * 1,2,4,5,6,7,8,8-OCTACHLORO-3a,4,7,7a-HEXAHYDRO-4,7-METHYLENE INDANE * OCTACHLORO-4,7-METHANOHYDROINDANE * OCTACHLORO-4,7-METHANOTETRAHYDROINDANE * 1,2,4,5,6,7,8,8-OCTACHLORO-4,7-METHANO-3a,4,7,7a-TETRAHYDROINDANE * 1,2,4,5,6,7,8,8-OCTACHLORO-3a,4,7,7a-TETRAHYDRO-4,7-METHANOINDAN * 1,2,4,5,6,7,8,8-OCTACHLORO-3a,4,7,7a-TETRAHYDRO-4,7-METHANOINDANE * 1,2,4,5,6,7,10;10-OCTACHLORO-4,7,8,9-TETRAHYDRO-4,7-METHYLENEINDANE * OCTA-KLOR * OCTATERR * ORTHO-KLOR * SD 5532 * SHELL SD-5532 * SYNKLOR * TAT CHLOR 4 * TOPICHLOR 20 * TOPICLOR * TOPICLOR 20 * TOXICHLOR * VELSICOL 1068
Chlorobenzilate	ACARABEN * AKAR * AKAR 338 * BENZILAN * CHLORBENZILAT * CHLOROBENZYLATE * COMPOUND 338 * 4,4'-DICHLOROBENZILIC ACID ETHYL ESTER * ENT 18,596 * ETHYL p,p'-

SUBSTANCE	SYNONYMS
Chlorobenzilate (Cont'd)	DICHLOROBENZILATE * ETHYL 4,4'-DICHLOROBENZILATE * ETHYL 4,4'-DICHLORODIPHENYL GLYCOLLATE * ETHYL 4,4'-DICHLOROPHENYL GLYCOLLATE * ETHYL ESTER of 4,4'-DICHLOROBENZILIC ACID * ETHYL-2-HYDROXY-2,2-BIS(4-CHLOROPHENYL)ACETATE * FOLBEX * FOLBEX SMOKE-STRIPS * G 338 * G23992 * GEIGY 338 * KOP-MITE * NCI-C00408 * NCI-C60413
Chloroform	CHLOROFORM (DOT) * FORMYL TRICHLORIDE * FREON 20 * METHANE TRICHLORIDE * METHANE, TRICHLORO * METHENYL TRICHLORIDE * METHENYL TRICHLORIDE * METHYL TRICHLORIDE * NCI-C02686 * R 20 * R 20 (REFRIGERANT) * TRICHLOROFORM * TRICHLOROMETHANE
Chromium & Certain Chromium Compounds	CHROME
Citrus Red No. 2	C.I. 12156 * C.I. SOLVENT RED 80 * 2,5-DIMETHOXYBENZENEAZO-beta-NAPHTHOL * 1-((2,5-DIMETHOXYPHENYL)AZO)-2-NAPHTHALENOL * 1-(2,5-DIMETHOXYPHENYLAZO)-2-NAPHTHOL * 2,5-DIMETHOXY-1-(PHENYLAZO)-2-NAPHTHOL * 1-(1-(2,5-DIMETHOXYPHENYL)AZO)-2-NAPHTHOL * 1-(2,5-DIMETHYLOXYPHENYLAZO)-2-NAPHTHOL
Coke Oven Emissions	NONE
p-Cresidine	3-AMINO-p-CRESOL METHYL ETHER * 1-AMINO-2-METHOXY-5-METHYLBENZENE * 3-AMINO-4-METHOXYTOLUENE * 2-AMINO-4-METHYLANISOLE * BENZENAMINE, 2-METHOXY-5-METHYL-(9CI) * CRESIDINE * KREZIDINE * 2-METHOXY-5-METHYLANILINE * 2-METHOXY-5-METHYLBENZENAMINE * 4-METHOXY-m-TOLUIDINE * 5-METHYL-o-ANISIDINE * NCI-C02982
Creosote	BRICK OIL * COAL TAR OIL * COAL TAR OIL (DOT) * CREOSOTE, COAL TAR (DOT) * CREOSOTE, from COAL TAR * CREOSOTE OIL * CREOSOTE OIL (DOT) * CREOSOTUM * CRESYLIC CREOSOTE * DEAD OIL (DOT) * HEAVY OIL * LIQUID PITCH OIL * NAPHTHALENE OIL * TAR OIL * WASH OIL
Cycasin	CYCAS REVOLUTA GLUCOSIDE * CYKAZINE * beta-D-GLUCOSYLOXYAZOXYMETHANE * beta-D-GLUCOSYLOXYAZOXYMETHASE * METHYLAZOXYMETHANOL-beta-D-GLUCOSIDE * (METHYL-ONN-AZOXY)-METHYL-beta-D-GLUCOPYRANOSIDE

ANCE	SYNONYMS
Cyclophosphamide	ASTA * B 518 * 2-(BIS(2-CHLOROETHYL)AMINO)-1-OXA-3-AZA-2-PHOSPHOCYCLOHEXANE 2-OXIDE MONOHYDRATE * 1-BIS(2-CHLOROETHYL)AMINO-1-OXO-2-AZA-5-OXAPHOSPHORIDINE MONOHYDRATE * 2-(BIS(2-CHLOROETHYL)AMINO)-2H-1,3,2-OXAZAPHOSPHORINE 2-OXIDE * (BIS(CHLORO-2-ETHYL)AMINO)2-2TETRAHYDRO-3,4,5,6-OXAZAPHOSPHORiNE-1,3,2-OXIDE-2 HYDRATE * 2-(BIS(2-CHLOROETHYL)AMINO)TETRAHYDRO(2H)-1,3,2-OXAZAPHOSPHORINE 2-OXIDE MONOHYDRATE * N,N-BIS(2-CHLOROETHYL)-N'-(3-HYDROXYPROPYL)PHOSPHORODIAMIDIC ACID intramol ESTER HYDRATE * BIS(2-CHLOROETHYL)PHOSPHORAMIDE CYCLIC PROPANOLAMIDE ESTER MONOHYDRATE * N,N-BIS(2-CHLOROETHYL)-N',O-PROPYLENEPHOSPHORIC ACID ESTER DIAMIDE * N,N-BIS(beta-CHLOROETHYL)-N',O-PROPYLENEPHOSPHORIC ACID ESTER AMIDE MONOHYDRATE * N,N-BIS(beta-CHLOROETHYL)-N',O-PROPYLENE PHOSPHORIC ACID ESTER DIAMIDE MONOHYDRATE * N,N-BIS(2-CHLOROETHYL)TETRAHYDRO-2H-1,3,2-OXAPHOSPHORIN-2-AMINE, 2-OXIDE MONOHYDRATE * N,N-BIS(beta-CHLOROETHYL)-N'O-TRIMETHYLENEPHOSPHORIC ACID ESTER DIAMIDE * CB-4564 * CLAFEN * CP * CTX * CY * CYCLIC N',O-PROPYLENE ESTER of N,N-BIS(2-CHLOROETHYL)PHOSPHORODIAMIDIC ACID MONOHYDRATE * CYCLOPHOSPHAMID * CYCLOPHOSPHAMIDUM * CYCLOPHOSPHAN * CYCLOPHOSPHANE * CYTOPHOSPHAN *
Cyclophosphamide (Cont'd)	CYTOXAN * 2-(DI(2-CHLOROETHYL)AMINO)-1-OXA-3-AZA-2-PHOSPHACYCLOHEXANE-2-OXIDE MONOHYDRATE * 2-(DI(2-CHLOROETHYL)-AMINO)2-OXIDE * N,N-DI(2-CHLOROETHYL)-AMINO-N,O-PROPYLENE PHOSPHORIC ACID ESTER DIAMIDE MONOHYDRATE * N,N-DI(2-CHLOROETHYL)-N,O-PROPYLENE-PHOSPHORIC ACID ESTER DIAMIDE * ENDOXAN * ENDOXANA * ENDOXAN-ASTA * ENDOXANE * ENDOXAN R * ENDUXAN * ENDOXANAL * GENOXAL * HEXADRIN * MITOXAN * NCI-C04900 * NSC 25271 * 2-H-1,3,2-OXAZAPHOSPHORINANE * 10-(3-(4-OXYAETHYLHOMOPIPERAZINO)PROPYL-(1, -2-CHLOR-4-AZAPHENTHIAZIN DIHYDROCHLORID * PHOSPHORODIAMIDIC ACID, N,N-BIS(2-CHLOROETHYL)-N'-(3-HYDROXYPROPYL -, intramol, ESTER * PROCYTOX * SEMDOXAN * SENDOXAN * SENDUXAN
Daunomycin	ACETYLADRIAMYCIN * CERUBIDIN * DAUNORUBICIN * DAUNORUBICINE * LEUKAEMOMYCIN C * NCI-C04693 * NSC-82151 * RP 13057 * 13057 R.P. * RUBIDOMYCINE * RUBOMYCIN C * RUBOMYCIN C 1 * STREPTOMYCES PEUCETIUS
Diallate	AVADEX * BIS(1-METHYLETHEL) CARBAMOTHIOIC ACID, S-(2,3-DICHLORO-2-PROPENYL)-ESTER * CARBAMOTHIOIC ACID, BIS(1-METHYLETHYL)-, S-(2,3-DICHLORO-2-PROPENYL) ESTER * CP 15,336 * DATC * 2,3-DCDT * DICHLORO-

SUBSTANCE	SYNONYMS
Diallate (Cont'd)	ALLYL DIISOPROPYLTHIOCARBAMATE * S-2,3-DICHLOROALLYL DIISOPROPYLTHIOCARBAMATE * 2,3-DICHLOROALLYL N, N-DIISOPROPYLTHIOLCARBAMATE * 2,3-DICHLORO-2-PROPENE-1-THIOL, DIISOPROPYLCARBAMATE * DIISOPROPYLTHIOCARBAMIC ACID, S-2,3-DICHLOROALLYL) ESTER * 2-PROPENE-1-THIOL, 2,3-DICHLORO-, DIISOPROPYLCARBAMATE
2,4-Diaminotoluene	3-AMINO-p-TOLUIDINE * 5-AMINO-o-TOLUIDINE * AZOGEN DEVELOPER H * BENZOFUR MT * C.I. 76035 * C.I. OXIDATION BASE 20 * C.I. OXIDATION BASE 200 * DEVELOPER 14 * DEVELOPER B * DEVELOPER DB * DEVELOPER DBJ * DEVELOPER H * DEVELOPER MC * DEVELOPER MT * DEVELOPER MT-CF * DEVELOPER MTD * DEVELOPER T * 1,3-DIAMINO-4-METHYLBENZENE * 2,4-DIAMINO-1-METHYLBENZENE * 2,4-DIAMINOTOLUENE * 2,4-DIAMINO-1-TOLUENE * 2,4-DIAMINOTOLUOL * EUCANINE GB * FOURAMINE * FOURAMINE J * FOURRINE 94 * FOURRINE M * META TOLUYLENE DIAMINE * 4-METHYL-1,3-BENZENEDIAMINE * 4-METHYL-m-PHENYLENEDIAMINE * MTD * NAKO TMT * NCI-C02302 * PELAGOL J * PELAGOL GREY J * PONTAMINE DEVELOPER TN * RENAL MD * TERTRAL G * 2,4-TOLAMINE * m-TOLUENEDIAMINE * 2,4-TOLUENEDIAMINE * m-TOLUYLENEDIAMINE * 2,4-TOLUYLENEDIAMINE * m-TOLYENEDIAMINE * TOLYLENE-2,4-DIAMINE * 2,4-TOLYLENEDIAMINE * 4-m-TOLYLENEDIAMINE * ZOBA GKE * ZOGEN DEVELOPER H

SUBSTANCE	SYNONYMS
Dibenz(a,h)acridine	DB(a,h)AC * 1,2,5,6-DIBENZACRIDINE * 1,2,5,6-DINAPHTHACRIDINE
Dibenz(a,j)acridine	7-AZADIBENZ(a,j)ANTHRACENE * 1,2,7,8-DIBENZACRIDINE * 1,2:7,8-DIBENZACRIDINE * 3,4,5,6-DIBENZACRIDINE * 3,4,6,7-DINAPHTHACRIDINE
Dibenz(a,h)anthracene	DB(a,h)A * 1,2,5,6-DBA * 1,2:5,6-DIBENZANTHRACENE * DIBENZO(a,h)ANTHRACENE
7H-Dibenzo(c,g)-carbazole	3,4,5,6-DIBENZCARBAZOL * 3,4,5,6-DIBENZCARBAZOLE * 3,4,5,6-DINAPHTHACARBAZOLE * 7H-DB(c,g)C
Dibenzo(a,e)pyrene	DB(a,e)P * DIBENZO(a,e)PYRENE * 1,2,4,5-DIBENZOPYRENE
Dibenzo(a,h)pyrene	DB(a,h)P * DIBENZO(b,def)CHRYSENE * 1,2,6,7-DIBENZOPYRENE * 3,4,8,9-DIBENZOPYRENE * 3,4,8,9-DIBENZPYRENE
Dibenzo(a,i)pyrene	DB(a,i)P * BENZO(rst)PENTAPHENE * DIBENZ(a,i)PYRENE * 1,2,7,8-DIBENZOPYRENE * 3,4:9,10-DIBENZOPYRENE * 3,4:9,10-DIBENZPYRENE

SUBSTANCE	SYNONYMS
1,2-Dibromo-3-chloropropane	BBC 12 • 1-CHLORO-2,3-DIBROMOPROPANE • 3-CHLORO-1-2-DIBROMOPROPANE • DBCP • DIBROMOCHLOROPROPANE • 1,2-DIBROOM-3-CHLOORPROPAAN (Dutch) • FUMAGON • FUMAZONE • FUMAZONE 86 • FUMAZONE 86E • NCI-C00500 • NEMABROM • NEMAFUME • NEMAGON • NEMAGON 20 • NEMAGON 90 • NEMAGON 206 • NEMAGON SOIL FUMIGANT • NEMANAX • NEMAPAZ • NEMASET • NEMATOCIDE • NEMATOX • OS 1897 • OXY DBCP • PROPANE, 1-CHLORO-2,3-DISBROMO- • SD 1897
1,2-Dibromoethane	BROMOFUME • CELMIDE • DBE • DIBROMOETHANE • sym-DIBROMOETHANE • alpha,beta-DIBROMOETHANE • 1,2-DIBROMOETHANE • 1,2-DIBROMOETHANE (DOT) • DOWFUME 40 • DOWFUME EDB • DOWFUME W-8 • DOWFUME W-85 • EDB • EDB-85 • E-D-BEE • ENT 15,349 • ETHYLENE BROMIDE • ETHYLENE DIBROMIDE • ETHYLENE DIBROMIDE (DOT) • FUMO-GAS • GLYCOL BROMIDE • GLYCOL DIBROMIDE • ISCOBROME D • KOPFUME • NCI-C00522 • NEPHIS • PESTMASTER • PESTMASTER EDB-85 • SOILBROM-40 • SOILBROM-85 • SOILBROME-85 • SOILBROM-90EC • SOILFUME • UNIFUME
3,3'-Dichlorobenzidine	C.I. 23060 • DCB • 4,4'-DIAMINO-3,3'-DICHLOROBIPHENYL • o,o'-DICHLOROBENZIDINE • 3,3'-DICHLOROBENZIDINE • DICHLOROBENZIDINE BASE • 3,3'-DICHLORO-4,4'-BIPHENYL-

SUBSTANCE	SYNONYMS
3,3'-Dichlorobenzidine (Cont'd)	DIAMINE • 3,3'DICHLOROBIPHENYL-4,4'-DIAMINE • 3,3'-DICHLORO-4,4'-DIAMINOBIPHENYL
Dichlorodiphenyltrichloroethane	ANOFEX • ARKOTINE • BENZENE, 1,1'-(2,2,2-TRICHLOROETHYLIDENE)BIS(4-CHLORO- • alpha,alpha-BIS(p-CHLOROPHENYL)-beta,beta,beta-TRICHLORETHANE • 2,2-BIS(p-CHLOROPHENYL)-1,1,1-TRICHLOROETHANE • CHLOROPHENOTHANE • DDT • p,p'-DDT • DDT (DOT) • DEDELO • DICHLORODIPHENYLTRICHLOROETHANE • p,p'-4,4'-DICHLORODIPHENYLTRICHLOROETHANE • DICHLORODIPHENYLTRICHLOROETHANE (DOT) • DICOPHANE • DIDIGAM • DIDIMAC • DIPHENYLTRICHLOROETHANE • ENT 1,506 • ESTONATE • GENITOX • GESAPON • GESAREX • GESAROL • GUESAROL • GYRON • IXODEX • KOPSOL • NCI-C00464 • NEOCID • PENTACHLORIN • PENTECH • PPZEIDAN • RUKSEAM • SANTOBANE • 1,1,1-TRICHLOR-2-2-BIS(4-CHLOR-PHENYL)-AETHAN (German) • TRICHLOROBIS(4-CHLOROPHENYL ETHANE • 1,1,1-TRICHLORO-2,2-BIS(p-CHLOROPHENYL)ETHANE • 1,1,1-TRICHLORO-2,2-DI(4-CHLOROPHENYL)-ETHANE • ZEIDANE • ZERDANE
1,2-Dichloroethane	1,2-BICHLOROETHANE • BORER SOL • BROCIDE • DESTRUXOL BORER-SOL • DICHLOREMULSION • 1,2-DICHLORETHANE • DI-CHLOR-MULSION • sym-DICHLOROETHANE • alpha,

SUBSTANCE	SYNONYMS
1,2-Dichloroethane (Cont'd)	beta-DICHLOROETHANE • 1,2-DICHLOROETHANE • DICHLOROETHYLENE • DUTCH LIQUID • DUTCH OL • EDC • ENT 1,656 • ETHANE DICHLORIDE • ETHYLENE CHLORIDE • ETHYLENE DICHLORIDE • ETHYLENE DICHLORIDE (DOT) • FREON 150 • GYCOL DICHLORIDE • NCI-C00511
Dieldrin	ALVIT • COMPOUND 497 • DIELDREX • DIELDRIN (DOT) • DIELDRITE • ENT 16,225 • HEOD • HEXACHLOROEPOXYOCTAHYDRO-endo-exo-DIMETHANONAPHTHALENE • ILLOXOL • NCI-C00124 • OCTALOX • PANORAM D-31 • QUINTOX
Diepoxybutane	1,1'-BI(ETHYLENE OXIDE) • BIOXIRAN • BIOXIRANE • 2,2'-BIOXIRANE • BUTADIENE DIOXIDE • 2,4-DIEPOXYBUTANE • 1,2:3,4-DIEPOXYBUTANE • DIOXYBUTADIENE • ERYTHRITOL ANHYDRIDE • THREITOL, 1,2:3,4-DIANHYDRO-
1,2-Diethylhydrazine	N,N'DIETHYLHYDRAZINE • sym-DIETHYLHYDRAZINE • HYDRAZOETHANE • HYDROAZOETHANE • SDEH
Diethylstilbestrol	ACNESTROL • ANTIGESTIL • BIO-DES • 3,4-BIS(p-HYDROXYPHENYL)3-3HEXENE • BUFON • CLIMATERINE • COMESTROL ESTROBENE • CYREN • CYREN A • DAWE'S DESTROL • DEB • DES • DESMA • DESTROL • DIBESTROL '2' PREMIX • DICORVIN • DI-ESTRYL • 4,4'-(1,2-DIETHYL-1,2-ETHENEDIYL)BIS-PHENOL • trans-4,4'-(1,2-

SUBSTANCE	SYNONYMS
Diethylstibestrol (Cont'd)	DIETHYL-1,2-ETHENEDIYLBISPHENOL • alpha-alpha'-DIETHYLSTILBENEDIOL • trans-alpha-alpha'-DIETHYL-4,4'-STILBENEDIOL • alpha, alpha'-DIETHYL-4,4'-STILBENEDIOL • trans-DIETHYLSTILBESTEROL • DIETHYLSTILBESTROL • trans-DIETHYLSTILBESTROL • DIETHANYLSTILBOESTEROL • trans-DIETHYLSTILBOESTEROL • 4,4'-DIHYDROXYDIETHYLSTILBENE • 4,4'-DIHYDROXY-alpha,beta-DIETHYLSTILBENE • 3,4-BIS(4,4'-DIHYDROXYPHENYL)HEX-3-ENE • DISTILBENE • DOMESTROL • ESTILBEN • ESTILBEN 'MCO' • ESTRIL • ESTROBENE • ESTROMENIN • ESTROSYN • FOLLIDIENE • FONATOL • GRAFESTROL • GYNOPHAM • 3-HEXENE,3,4-BIS(p-HYDROXYPHENYL)- • HIBESTROL • IDROESTRIL • ISCOVESCO • MENOSTILBEEN • MICROEST • MILESTROL • NEO-OESTRANOL 1 • NSC-3070 • OEKOLP • OESTROGEN • OESTROGENINE • OESTROL VETAG • OESTROMENIN • OESTROMENSIL • OESTROMENSYL • OESTROMIENIN • OESTROMON • PABESTROL • PALESTROL • PERCUTATRINE OESTROGENIQUE ISCOVESCO • PROTECTONA • RUMESTROL 1 • RUMESTROL 2 • SEDESTRAN • SERRAL • SEXOCRETIN • SIBOL • SINTEROL • STIBILIUM • STIL • 4,4'-STILBENEDIOL,2,2'-DIETHY- • STILBESTROL • STILBESTROL, DIETHYL- • STILBETIN • STILBOEFRAL • STILBOESTROFORM • STILBOESTROL • STILBOFOLLIN • STILBOL • STILKAP • STILROL • SYNESTRIN • SYNTHOESTRIN • SYNTHOFOLIN • SYNTOFOLIN • TAMPOVAGAN STILBOESTROL • TYLOSTERONE • VAGESTROL

ANCE	SYNONYMS
3,3'-Dimethoxybenzidine	ACETAMINE DIAZO BLACK RD • ACETAMINE DIAZO NAVY RD • AMACEL DEVELOPED NAVY SD • AZOENE FAST BLUE BASE • AZOENE FAST BLUE SALT • AZOFIX BLUE B SALT • AZOGENE FAST BLUE B • AZOGENE FAST BLUE B SALT • BLUE BASE IRGA B • BLUE BASE NB • BLUE BN BASE • BLUE BN SALT • BLUE SALT NB • BRENTAMINE FAST BLUE B BASE • BRENTAMINE FAST BLUE B SALT • CELLITAZOL B • CELLITAZOL BN • CIBACETE DIAZO NAVY BLUE 2B • C.I. 24110 • C.I. AZOIC DIAZO COMPONENT 48 • C.I. AZOIC DIAZO COMPONENT 48, FAST BLUE B SALT • C.I. DISPERSE BLACK 6 • DIACELLITON FAST GREY G • DIACEL NAVY DC • DIANISIDINE • o-DIANISIDINE • O,O'-DIANISIDINE • DIATO BLUE BASE B • DIATO BLUE SALT B • DIAZO FAST BLUE B • 3,3'-DIMETHOXYBENZIDINE • FAST BLUE B BASE • FAST BLUE BN SALT • FAST BLUE DSC BASE • FAST BLUE DS SALT • FAST BLUE SALT B • FAST BLUE SALT BN • HILTONIL FAST BLUE B BASE • HILTOSAL FAST BLUE B SALT • HINDASOL BLUE B SALT • KAKO BLUE B SALT • KAYAKU BLUE B BASE • KAYAKU BLUE B SALT • LAKE BLUE B BASE • MEISEI TERYL DIAZO BLUE HR • MITSUI BLUE B BASE • MITSUI BLUE B SALT • NAPHTHANIL BLUE B BASE • NATASOL BLUE B SALT • NEUTROSEL NAVY BN • SANYO FAST BLUE SALT B • SETACYL DIAZO NAVY R • SPECTROLENE BLUE B

SUBSTANCE	SYNONYMS
4-Dimethylaminoazobenzene	ATUL FAST YELLOW R • AZOBENZENE, p-DIMETHYLAMINE- • BENZENAMINE, N,N-DIMETHYL-4-(PHENYLAZO)- (9CI) • BENZENEAZODIMETHYLANILINE • BRILLIANT FAST OIL YELLOW • BRILLIANT FAST SPIRIT YELLOW • BRILLIANT FAST YELLOW • BRILLIANT OIL YELLOW • BUTTER YELLOW • CERASINE YELLOW GG • C.I. 11020 • C.I. SOLVENT YELLOW 2 • DAB • DAB (carcinogen) • DIMETHYLAMINOAZOBENZENE • N,N-DIMETHYL-4-AMINOAZOBENZENE • N,N-DIMETHYL-p-AMINOAZOBENZENE • p-DIMETHYLAMINOAZOBENZENE • 4-(N,N-DIMETHLYAMINO)AZOBENZENE • DIMETHYLAMINOAZOBENZOL • 4-DIMETHYLAMINOAZOBENZOL • 4-DIMETHYLAMINOPHENYLAZOBENZENE • N,N-DIMETHYL-p-AZOANILINE • N,N-DIMETHYL-p-PHENYLAZOANILINE • N,N-DIMETHYL-4-(PHENYLAZO)BENZAMINE • N,N-DIMETHYL-4-(PHENYLAZO)BENZENAMINE • DIMETHYL YELLOW • DIMETHYL YELLOW ANALAR • DIMETHYL YELLOW N,N-DIMETHYLANILINE • DMAB • ENIAL YELLOW 2G • FAST OIL YELLOW B • FAST YELLOW • FAT YELLOW • FAT YELLOW A • FAT YELLOW AD OO • FAT YELLOW ES • FAT YELLOW ES EXTRA • FAT YELLOW EXTRA CONC. • FAT YELLOW R • FAT YELLOW R (8186) • GRASAL BRILLIANT YELLOW • METHYL YELLOW • OIL YELLOW • OIL YELLOW II • OIL YELLOW 20 • OIL YELLOW 2625 • OIL YELLOW 7463 • OIL YELLOW BB • OIL YELLOW D • OIL YELLOW DN • OIL YELLOW FF • OIL YELLOW FN • OIL YELLOW G • OIL YELLOW 2G • OIL

SUBSTANCE	SYNONYMS
4-Dimethylaminoazobenzene (Cont'd)	YELLOW G-2 • OIL YELLOW GG • OIL YELLOW GR • OIL YELLOW N • OIL YELLOW PEL • OLEAL YELLOW 2G • ORGANOL YELLOW ADM • ORIENT OIL YELLOW GG • P.D.A.B. • PETROL YELLOW WT • RESINOL YELLOW GR • RESOFORM YELLOW GGA • SILOTRAS YELLOW 12G • SOMALIA YELLOW A • STEAR YELLOW JB • SUDAN GG • SUDAN YELLOW • SUDAN YELLOW GG • SUDAN YELLOW GGA • TOYO OIL YELLOW G • USAF EK-338 • WAXOLINE YELLOW AD • WAXOLINE YELLOW ADS • YELLOW G SOLUBLE in GREASE
7,12-Dimethylbenz(a)anthracene	DBA • 9,10-DIMETHYL-1,2-BENZANTHRACENE • DIMETHYLBENZANTHRENE • 1,4-DIMETHYL-2,3-BENZPHENANTHRENE • DMA • 7,12-DMBA • NCI-C03918
Dimethylcarbamoyl Chloride	CARBAMYL CHLORIDE, N,N-DIMETHYL- • CHLOROFORMIC ACID DIMETHYLAMIDE • DDC • (DIMETHYLAMINO)CARBONYL CHLORIDE • DIMETHYLCARBAMIC ACID CHLORIDE • DIMETHYLCARBAMIC CHLORIDE • DIMETHYLCARBAMIDOYL CHLORIDE • DIMETHYLCARBAMOYL CHLORIDE • N,N-DIMETHYLCARBAMOYL CHLORIDE • DIMETHYLCARBAMYL CHLORIDE • N,N-DIMETHYLCARBAMYL CHLORIE • DMCC • TL 389
1,1-Dimethylhydrazine	DIMAZINE • 1,1-DIMETHYLHYDRAZINE • DIMETHYLHYDRAZINE • asymmetric DIMETHYLHYDRAZINE • N,N-DIMETHYLHYDRAZINE • uns-

SUBSTANCE	SYNONYMS
1,1-Dimethylhydrazine (Cont'd)	DIMETHYLHYDRAZINE • unsym-DIMETHYLHYDRAZINE • 1,1-DIMETHYL HYDRAZINE • DIMETHYLHYDRAZINE UNSYMMETRICAL (DOT) • UDMH
1,2-Dimethylhydrazine	N,N'-DIMETHYLHYDRAZINE • 1,2-DIMETHYLHYDRAZINE • sym-DIMETHYLHYDRAZINE • DMH • HYDRAZOMETHANE • SDMH
Dimethyl Sulfate	DIMETHYL MONOSULFATE • DIMETHYL SULFATE (DOT) • DIMETHYL SULPHATE • DMS (METHYL SULFATE) • DIMETHYLOWY METHYL SULFATE • SULFATE DE
2,4-Dinitrotoluene	NONE
1,4-Dioxane	DIETHYLENE DIOXIDE • 1,4-DIETHYLENE DIOXIDE • DIETHYLENE ETHER • DI(ETHYLENE OXIDE) • 1,4-DIOXACYCLOHEXANE • DIOKAN • DIOXAN • DIOXANE • 1,4-DIOXANE • DIOXANE-1,4 • DIOXANE (DOT) • p-DIOXIN, TETRAHYDRO- • DIOXYETHYLENE ETHER • GLYCOL ETHYLENE ETHER • NCI-C03689 • TETRAHYDRO-p-DIOXIN • TETRAHYDRO-1,4-DIOXIN
Epichlorohydrin	1-CHLORO-2,3-EPOXYPROPANE • 3-CHLORO-1,2-EPOXYPROPANE • (CHLOROMETHYL)ETHYLENE OXIDE • CHLOROMETHYLOXIRANE • 2-(CHLOROMETHYL)OXIRANE • CHLOROPROPYL-

SUBSTANCE	SYNONYMS
...ichlorohydrin (Cont'd)	ENE OXIDE * gamma-CHLOROPROPYLENE OXIDE * 3-CHLORO-1,2-PROPYLENE OXIDE * ECH * EPICHLOROHYDRIN (DOT) * alpha- EPICHLOROHYDRIN * (DL)-alpha-EPICHLOROHYDRIN * EPICHLOROPHYDRIN * 1,2-EPOXY-3-CHLOROPROPANE * 2,3-EPOXYPROPYL CHLORIDE * GLYCEROL EPICHLORHYDRIN * OXIRANE, (CHLOROMETHYL) * OXIRANE, 2-(CHLOROMETHYL) * SKEKhG
Ethylene Bis Dithiocarbamate	CHEM BAM * DISODIUM ETHYLENEBIS(DITHIOCARBAMATE) * DISODIUM ETHYLENE-1,2-BIS-DITHIOCARBAMATE * DITHANE A 40 * DITHANE D-14 * DSE * ETHYLENEBIS(DITHIOCARBAMATE), DISODIUM SALT * ETHYLENEBIS(DITHIOCARBAMIC ACID) DISODIUM SALT * NABAM * NABASAN * PARATE * PARZATE LIQUID * SPRING-BAK
Ethylene Oxide	ANPROLENE * DIHYDROOXIRENE * DIMETHYLENE OXIDE * E.O. 1,2-EPOXYETHANE * ETHYLENE OXIDE (DOT) * NCI-C50088 * OXACYCLOPROPANE * OXANE * OXIDOETHANE * alpha, beta-OXIDOETHANE * OXIRAN * OXIRANE * OXIRENE, DIHYDRO-
Ethylene Thiourea	4,5-DIHYDROIMIDAZOLE 2(3H)-THIONE * 4,5-DIHYDRO- * N,N'-ETHYLENETHIOUREA * 1,3-ETHYLENE-2-THIOUREA * ETU * (MIDAZOLE-2(3H)-THIONE, 2-IMIDAZOLIDINETHIONE *

SUBSTANCE	SYNONYMS
Ethylene Thiourea (Cont'd)	N-22 * NA-22-D * NCI-C03372 * PENNAC CRA * SODIUM-22 NEOPRENE ACCELERATOR * 2-THIOL-DIHYDROGLYOXALINE * UREA, 1,3-ETHYLENE-2-THIO- * WARECURE C
Ethylenimine	AMINOETHYLENE * AZACYCLOPROPANE * AZIRIDINE * AZIRANE * 1H-AZIRINE, DIHYDRO * DIHYDROAZIRENE * DIHYDRO-1H-AZIRINE * DIMETHYLENEIMINE * DIMETHYLENIMINE * EI * ETHYLENEIMINE * ETHYLIMINE * TL 337
Ethyl Methanesulfonate	ETHYL ESTER of METHANESULFONIC ACID * ETHYL ESTER of METHANESULPHONIC ACID * ETHYL ESTER of METHYLSUFONIC ACID * ETHYL ESTER of METHYLSULPHONIC ACID * EMS * ETHYL METHANESULFONATE * ETHYL METHANE SULPHONATE * ETHYL METHANSULPHONATE * HALF-MYLERAN * METHANESULPHONIC ACID ETHYL ESTER * METHYLSULFONIC ACID, ETHYL ESTER * NSC 26805
Formaldehyde	BFV * FANNOFORM * FORMALDEHYDE, as FORMALIN solution (DOT) * FORMALIN * FORMALITH * FORMIC ALDEHYDE * FORMOL * FYDE * HOCH * KARSAN * METHANAL * METHYL ALDEHYDE * METHLENE OXIDE * NCI-C02799 * OXOMETHANE * OXYMETHYLENE

SUBSTANCE	SYNONYMS
Glycidaldehyde	EPIHYDRINALDEHYDE * EPIHYDRINE ALDEHYDE * 2,3-EPOXYPROPANAL * 2,3-EPOXY-1-PROPANAL * 2,3-EPOXYPROPIONALDEHYDE * GLYCIDAL * OXIRANE-CARBOXALDEHYDE * PROPIONALDEHYDE, 2,3-EPOXY-
Hematite	BLOOD STONE * HAEMATITE * IRON ORE * RED IRON ORE
Heptachlor	AGROCERES * 3-CHLOROCHLORDENE * DICYCLOPENTADIENE, 3,4,5,6,7,8,8a-HEPTACHLORO- * DRINOX * DRINOX H-34 * E 3314 * ENT 15,152 * GPKh * H * 3,4,5,6,7,8,8-HEPTACHLORODICYCLOPENTADIENE * 3,4,5,6,7,8,8a-HEPTACHLORODICYCLOPENTADIENE * 1,4,5,6,7,8,8-HEPTACHLORO-3a,4,7,7a-TETRAHYDRO-4,7-ENDOMETHANOINDENE * 1,4,5,6,7,8,8-HEPTACHLORO-3a,4,7,7a-TETRAHYDRO-4,7-METHANOINDENE * 1(3a),4,5,6,7,8,8-HEPTACHLORO-3a(1),4,7,7a-TETRAHYDRO-4,7-METHANOINDENE * 3a,4,5,6,7,8,8-HEPTACHLORO-3a,4,7,7a-TETRAHYDRO-4,7-METHANOINDENE * 1,4,5,6,7,8,8-HEPTACHLORO-3a,4,7,7a-TETRAHYDRO-4,7-METHANOL-1H-INDENE * 1,4,5,6,7,8,8-HEPTACHLORO-3a,4,7,7,7a-TETRAHYDRO-4-7-METHYLENE INDENE * 1,4,5,6,7,10,10-HEPTACHLORO-4,7,8,9-TETRAHYDRO-4,7-METHYLENEINDENE * 1,4,5,6,7,10,10-HEPTACHLORO-4,7,8,9-TETRAHYDRO-4,7-ENDOMETHYLENEINDENE * HEPTAGRAN * HEPTAMUL * NCI-C00180 * RHODIACHLOR * VELSICOL 104 * VELSICOL HEPTACHLOR

SUBSTANCE	SYNONYMS
Hexachlorobenzene	AMATIN * ANTICARIE * BUNT-CURE * BUNT-NO-MORE * CO-OP HEXA * GRANOX NM * HCB * HEXA C.B. * HEXACHLOROBENZENE * JULIN'S CARBON CHLORIDE * NO BUNT * NO BUNT 40 * NO BUNT 80 * NO BUNT LIQUID * PENTACHLOROPHENYL CHLORIDE * PERCHLOROBENZENE * PHENYL PERCHLORYL * SANOCIDE * SMUT-GO * SNIECIOTOX
Hexachlorobutadiene	C-46 * DOLEN-PUR * GP-40-66:120 * HCBD * HEXACHLORBUTADIENE * HEXACHLOROBUTADIENE * 1,1,2,3,4,4-HEXACHLORO-1,3,BUTADIENE * PERCHLOROBUTADIENE
Hexachloroethane	AVLOTHANE * CARBON HEXACHLORIDE * DISTOKAL * DISTOPAN * DISTOPIN * EGITOL * ETHANE HEXACHLORIDE * ETHYLENE HEXACHLORIDE * FALKITOL * FASCIOLIN * HEXACHLOROETHANE * HEXACHLOROETHANE (DOT) * 1,1,1,2,2-HEXACHLOROETHANE * HEXACHLOROETHYLENE * MOTTENHEXE * NCI-C04604 * PERCHLOROETHANE * PHENOHEP
Hydrazine	DIAMIDE * DIAMINE * HYDRAZINE BASE * HYDRAZINE, ANHYDROUS (DOT)
Hydrazobenzene	BENZENE, HYDRAZODI- * N,N'-BIANILINE * 1,2-DIPHENYLHYDRAZINE (9CI) * (sym)-DIPHENYLHYDRAZINE * HYDRAZINE, 1,2-DIPHENYL- * NCI-C01854

SUBSTANCE	SYNONYMS
Indeno(1,2,3-cd)pyrene	IP * 2,3-PHENYLENEPYRENE * 2,3-o-PHENYLENEPYRENE
Iron Dextran	DEXTRAN IRON COMPLEX * IMFERON * IRON DEXTRAN INJECTION * IRONORM INJECTION * URSOFERRAN
Isopropyl Alcohol Manufacturing	AVANTINE * DIMETHYLCARBINOL * ISOHOL * ISOPROPANOL * LUTOSOL * PETROHOL * PRO * PROPAN-2-OL * 2-PROPANOL * sec-PROPYL ALCOHOL
Kepone	CIBA 8514 * CHLORDECONE * COMPOUND 1189 * 1,2,3,5,6,7,8,9,10,10-DECACHLORO(5.2.1.0 (sup 2,6)-0(sup 3,9).0(sup 5,8))DECANO-4-ONE * DECACHLOROKETONE * DECACHLORO-1,3,4-METHENO-2H-CYCLOBUTA(cd)PENTALEN-2-ONE * DECACHLOROOCTAHYDRO-1,3,4-METHENO-2H-CYCLOBUTA(cd)PENTALEN-2-ONE * 1,1a,3,3a,4,5,5a5b,6-DECACHLOROOCTAHYDRO-1,3-4-METHENO-2H-CYCLOBUTA(cd)PENTALEN-2-ONE * DECACHLOROPENTACYCLO(5.2.1.9(sup 2,6) o(sup 3,9) o(sup 5,8))DECAN-4-ONE * DECACHLOROPENTACYCLO(5.3.0.0(sup 2,6).0(sup 4,10) 0(sup 5,9)DECAN-3-ONE * DECACHLOROTETRACYCLODECANONE * DECACHLOROTETRAHYDRO-4,7-METHANOINDENEONE * ENT 16,391 * GC 1189 * GENERAL CHEMICALS 1189 * KEPONE-2-ONE, DECACHLOROOCTAHYDRO- * MEREX * NCI-C00191
Lasiocarpine	HELIOTRIDINE ester with LASIOCARPUM and ANGELIC ACID * NCI-C01478
Lead Acetate	DIBASIC LEAD ACETATE * LEAD ACETATE (Pb(Ac)2) * LEAD ACETATE (Pb(O2C2H3)2) * LEAD(2+) ACETATE * LEAD(II) ACETATE * LEAI DIACETATE * LEAD DIBASIC ACETATE * NORMAL LEAD ACETATE * PLUMBOUS ACETATE * SALT OF SATURN * SUGAR OF LEAD
Lead Phosphate	C.I. 77622 * LEAD ORTHOPHOSPHATE * LEAD PHOSPHATE (3:2) * LEAD(2+) PHOSPHATE * NORMAL LEAD ORTHOPHOSPHATE * PERLEX PASTE 500 * PERLEX PASTE 600A * PHOSPHOR ACID, LEAD(2+) SALT (2:3) * PLUMBOUS PHOPHATE * TRILEAD PHOSPHATE
Lindane	AALINDAN * AFICIDE * AGRISOL G-20 * AGRCIDE * AGROCIDE 2 * AGROCIDE 6G * AGROCIDE 7 * AGROCIDE III * AGROCID WP * AGRONEXIT * AMEISENMITTEL MERCK * AMEI SENATOD * APARASIN * APHTIRIA * APLIDAL ARBITEX * BBH * BEN-HEX * BENTOX 10 * BEN ZENE HEXACHLORIDE-gamma-isomer * gamma BENZENE HEXACHLORIDE * BEXOL * BHC * gamma-BHC * CELANEX * CHLORESENE * CODECHINE * DBH * DETMOL-EXTRAKT * DETOX 25 * DEVORAN * DOL GRANULE * DRILL TOX-SPEZIAL AGLUKON * ENT 7,796 * ENTOMOXAN * EXAGAMA * FORLIN * GALLOGAMA

SUBSTANCE	SYNONYMS
Lindane (Cont'd)	* GAMACID * GAMAPHEX * GAMMAHEXA * GAMMAHEXANE * GAMMALIN * GAMMALIN 20 * GAMMATERR * GAMMEX * GAMMEXANE * GAMMOPAZ * GEXANE * HCCH * HCH * gamma-HCH * HECLOTOX * HEXA * HEXACHLORAN * gamma-HEXACHLORAN * HEXACHLORANE * gamma-HEXACHLORANE * gamma-HEXACHLOROBENZENE * 1-alpha,2-alpha,3-beta,4-alpha,5-alpha,6-beta-HEXACHLOROCYCLOHEXANE * gamma-HEXACHLOROCYCLOHEXANE * 1,2,3,4,5,6-HEXACHLOROCYCLOHEXANE, gamma-ISOMER * gamma-1,2,3,4,5,6-HEXACHLOROCYCLOHEXANE * HEXATOX * HEXAVERM * HEXICIDE * HEXYCLAN * HGI * HORTEX * INEXIT * ISOTOX * JACUTIN * KOKOTINE * KWELL * LENDINE * LENTOX * LIDENAL * LINDAFOR * LINDAGAM * LINDAGRAIN * LINDAGRANOX * LINDANE (DOT) * gamma-LINDANE * LINDAPOUDRE * LINDATOX * LINDOSEP * LINTOX * LOREXANE * MILBOL 49 * MSZYCOL * NCI-C00204 * NEO-SCABICIDOL * NEXEN FB * NEXIT * NEXIT-STARK * NEXOL-E * NICOCHLORAN * NOVIGAM * OMNITOX * OVADZIAK * OWADZIAK PEDRACZAK * PFLANZOL * QUELLADA * SANG gamma * SILVANO * SPRITZ-RAPIDIN * SPRUEHPFLANZOL * STREUNEX * TAP 85 * TRI-6 * VITON
Melphalan	ALKERAN * AT-290 * L-3-(p-(BIS(2-CHLOROETHYL)AMINO)PHENYL)ALANINE * p-N-BIS(2-CHLOROETHYL)AMINO-L-PHENLALANINE * 3-(p-(BIS(2-CHLOROETHYL)AMINO)PHENYL)-L-
Melphalan (Cont'd)	ALANINE * 4-(BIS(2-CHLOROETHYL)AMINO)-L-PHENYLALANINE * CB 3025 * 3025 C.B. * p-DI-(2-CHLOROETHYL)AMINO)L-PHENYLALANINE * 3-6-(DI(2-CHLOROETHYL)AMINO)-PHENYL-L-ALANINE * MELFALAN * NCI-C04853 * NSC-8806 * L-PAM * PHENYLALANINE MUSTARD * L-PHENYLALANINE MUSTARD * PRENYLALANINE NITROGEN MUSTARD * L-SARCOLYSIN * p-L-SARCOLYSIN * SARCOLYSINE * L-SARCOLYSINE * SARKOLYSIN * L-SARKOLYSIN * SK-15673
Methapyrilene	A 3322 * AH-42 * 2-((2-(DIMETHYLAMINO) ETHYL)-2-THENYLAMINO)PYRIDINE * N,N-DIMETHYL-N'-PYRID-2-YL-N'-2-THENYLETHYL-ENEDIAMINE * HISTADYL * LULAMIN * NCI-C55550 * PARADORMALENE * N-(alpha-PYRIDYL)-N-(alpha-THENYL)-N',N'-DIMETHYL-ENEDIAMINE * PYRINSTAB * PYRINISTOL * RESTON * RESTRYL * SEMIKON * SLEEPWELL * TENALIN * THENYLENE * THENYLPYRAMINE * THIONYLAN
3-Methyl-cholanthrene	BENZ(j)ACEANTHRYLENE, 1,2-DIHYDRO-3-METHYL- * MC * 3-MC * 20-MC * MCA * 3-MCA * METHYLCHOLANTHRENE * 20-METHYL-CHOLANTHRENE
4-4'-Methylenebis(2-Chloroaniline)	ANILINE, 4,4'-METHYLENEBIS(2-CHLORO- * BIS AMINE * CL-MDA * CURALIN M * CURENE 442 * CYANASET * DACPM * (DI(4-AMINO-3-CHLOROPHENYL)METHANE * DI-(4-AMINO-3-CLORO-

SUBSTANCE	SYNONYMS
4,4'-Methylenebis(2-Chloroaniline) (Cont'd)	FENIL)METANO (Italian) • 4,4'-DIAMINO-3,3'-DICHLORODIPHENYLMETHANE • 3,3'-DICHLORO-4,4'-DIAMINODIPHENYLMETHANE • MBOCA • 4,4'-METHYLENE(BIS)-CHLOROANILINE • METHYLENE 4,4'-BIS(o-CHLOROANILINE) • p,p'-METHYLENEBIS(alpha-CHLOROANILINE) • 4,4'-METHYLENEBIS(o-CHLOROANILINE) • p,p'-METHYLENEBIS(o-CHLOROANILINE) • 4,4'-METHYLENEBIS-2-CHLOROBENZENAMINE • MOCA
Methyl Iodide	HALON 10001 • IODOMETHANE
Methyl Methanesulfonate	as-DIMETHYL SULPHITE • METHANESULPHONIC ACID METHYL ESTER • METHYL ESTER of METHANESULFONIC ACID • METHYL ESTER of METHANESULPHONIC ACID • METHYL MESYLATE • METHYL METHANESULFONATE • METHYL METHANESULPHONATE • METHYL METHANSULPHONAE • MMS • NSC-50256
N-Methyl-N'-nitro-N-nitrosoguanidine	N-METHYL-N'-NITRO-N-NITROSOGUANIDINE • 1-METHYL-3-NITRO-NITROSOGUANIDINE • MNG • MNNG • N'-NITRO-N-NITROSO-N METHYLGUANIDINE
Methylthiouracil	ALKIRON • ANTIBASON • BASECIL • BASETHYRIN • 2,3-DIHYDRO-6-METHYL-2-THIOXO-4(1H)-PYRIMIDINONE • 2-MERCAPTO-6-METHYLPYRIMID-4-ONE • 2-MERCAPTO-4-

SUBSTANCE	SYNONYMS
Methylthiouracil (Cont'd)	HYDROXY-6-METHYLPYRIMIDINE • 2-MERCAPTO-6-METHYL-4-PYRIMIDONE • METACIL • METHACIL • METHIACIL • METHICIL • METHIOCIL • 6-METHYL-2-THIO-2,4-(1H3H)-PYRIMIDINDIONE • 6-METHYLTHIOURACIL • 4-METHYL-2-THIOURACIL • 6-METHYL-2-THIOURACIL • 4-METHYLURACIL • MTU • MURACIL • ORCANO • PROSTRUMYL • STRUMACIL • THIMECIL • THIOMECIL • 2-THIO-6-METHYL-1,3-PYRIMIDIN-4-ONE • 6-THIO-4-METHYLURACIL • THIOMID • 2-THIO-4-OXO-6-METHYL-1,3-PYRIMIDINE • THIORYL • THIOTHYRON • THIURYL • THYRECSTAT • THYREOSTAT I • TIOTIRON • THYRIL • USAF EK-6454
Mirex	BICHLORENDO • CG-1283 • CYCLOPENTADIENE, HEXACHLORO-, DIMER • DECANE,PERCHLOROPENTACYCLO- • DECHLORANE • DECHLORANE 515 • DECHLORANE 4070 • DECHLORANE PLUS • DECHLORANE PLUS 515 • DODECACHLOROOCTAHYDRO-1,3,4-METHENO-2H-CYCLOBUTA(c,d)PENTALENE • DODECACHLOROPENTACYCLODECANE • DODECACHLOROPENTACYCLO(3,2,2,0(sup 2,6),0(sup 3,9),(sup 5,10))DECANE • ENT 25,719 • FERRIAMICIDE • GC 1283 • HEXACHLOROCYCLOPENTADIENE DIMER • 1,2,3,4,5,5-HEXACHLORO-1,3-CYCLOPENTADIENE DIMER • HRS 1276 • 1,3,4-METHENO-1H-CYCLOBUTA(cd)PENTALENE, DODECACHLOROOCTAHYDRO- • NCI-C06428 • PERCHLORODIHOMOCUBANE • PERCHLORO-

SUBSTANCE	SYNONYMS
Mirex (Cont'd)	PENTACYCLODECANE • PERCHLOROPENTACYCLO(5.2.1.0(sup 2,6),0(sup 3,9),0(sup 5,8))DECANE
Mitomycin C	AMETYCIN • MIT-C • MITOMYCIN • MITOMYCINUM • MMC • MUTAMYCIN(MITOMYCIN for INJECTION) • MYTOMYCIN, NSC 26980 • NCI-C04706
Mustard Gas	BIS(beta-CHLOROETHYL)SULFIDE • BIS(2-CHLOROETHYL)SULFIDE • BIS-2-CHLOROETHYL)SULPHIDE • 1-CHLORO-2-(beta-CHLOROETHYLTHIO)ETHANE • 2,2'-DICHLORODIETHYL SULFIDE • DI-2-CHLOROETHYL SULFIDE • beta-beta'-DICHLOROETHYL SULFIDE • beta-beta-DICHLOROETHYL-SULPHIDE • 2,2'-DICHLOROETHYL SULPHIDE • DISTILLED MUSTARD • H • HD • KAMPSTOFF 'LOST' • MUSTARD HD • MUSTARD, SULFUR • MUSTARD VAPOR • SCHWEFELLOST • S-LOST • SULFUR MUSTARD GAS • SULFUR MUSTARD • S MUSTARD • SULPHUR MUSTARD • SULPHUR MUSTARD GAS • 1,1'-THIOBIS(2-CHLOROETHANE • YELLOW CROSS LIQUID • YPERITE
1-Naphthylamine	1-AMINONAPHTHALENE • C.I. AZOIC DIAZO COMPONENT 114 • FAST GARNET BASE • FAST GARNET BASE B • NAPHTHALIDAM • NAPHTHALIDINE • alpha-NAPHTHYLAMINE

SUBSTANCE	SYNONYMS
2-Naphthylamine	2-AMINONAPHTHALENE • C.I. 3270 • FAST SCARLET BASE B • NA • 2-NAPHTHALAMINE • 2-NAPHTHALENAMINE • beta-NAPHTHYLAMINE • 6-NAPHTHYLAMINE • 2-NAPHTHYLAMINE MUSTARD • USAF CB-22
Nickel & Certain Nickel Compounds	C.I. 77775 • Ni 270 • NICKEL 270 • NICKEL CATALYST, WET (DOT) • NICKEL SPONGE • Ni 0901-S • Ni 4303T • NP 2 • PULVERIZED NICKEL • RANEY ALLOY • RANEY NICKEL • RCH 55/5
Nitrogen-Mustard and its hydrochloride	BIS(2-CHLOROETHYL)METHYLAMINE HYDROCHLORIDE • CARYOLSINE • CHLORAMIN • CHLORAMINE • CHLORETHAMINE • CHLORETHAZINE • 2-CHLORO-N-(2-CHLOROETHYL)-N-METHYLETHANAMINE HYROCHLORIDE • DICHLOREN • beta,beta'-DICHLORODIETHYL-N-METHYLAMINE HYDROCHLORIDE • DI(2-CHLOROETHYL)METHYLAMINE HYDROCHLORIDE • 2,2'-DICHLORO-N-METHYLDIETHYLAMINE HYDROCHLORIDE • DIMITAN • EMBICHIN • EMBIKHINE • ERASOL • HN2.HCl • HN2 HYDROCHLORIDE • N-LOST • MBA HYDROCHLORIDE • MEBICHLORAMINE • MECHLORETHAMINE HYDROCHLORIDE • N-METHYL-BIS-beta-CHLORETHYLAMINE HYDROCHLORIDE • METHYLBIS (2-CHLOROETHYL)AMINE HYDROCHLORIDE • N-METHYL-2,2'-DICHLORODIETHYLAMINE HYDROCHLORIDE • N-METHYL-DI-2-CHLOROETHYLAMINE HYDROCHLORIDE • METHYLDI (beta-CHLOROETHYL)AMINE HYDROCHLORIDE

SUBSTANCE	SYNONYMS
Nitrogen-Mustard and its hydrochloride (Cont'd)	* METHYLDI(2-CHLOROETHYL)AMINE HYDROCHLORIDE * MITOXINE * MUSTINE HYDROCHLOR * MUSTARGEN HYDROCHLORIDE * MUSTINE HYDROCHLORIDE * NCI-C56382 * NITOL 'TAKEDA' * NITROGRANULOGEN * NSC-762 HYDROCHLORIDE
5-Nitro-o-toluidine	C.I. 37105 * C.I. AZOIC DIAZO COMPONENT 12 * FAST SCARLET BASE G * 6-METHYL-3-NITRO-ANILINE * 2-METHYL-5-NITRO-BENZENEAMINE * NCI-C01843 * 4-NITRO-2-AMINOTOLUENE
4-Nitroquinoline-1-oxide	4-NITROQUINOLINE-N-OXIDE * 4-NQO
N-Nitrosodi-ethanolamine	BIS(beta-HYDROXYETHYL)NITROSAMINE * DI-ETHANOLNITROSOAMINE * DIETHYLAMINE, 2,2'-DIHYDROXY-N-NITROSO- * NCI-C55583 * 2,2'-IMINODI-N-NITROSOETHANOL * NDEA * NDELA * N-NITROSOAMINODIETHANOL * N-NITROSOBIS(2-HYDROXYETHYL)AMINE * 2,2'-(NITROSOIMINO)BISETHANOL * NITROSOIMINO DIETHANOL
N-Nitrosodi-ethylamine	DANA * DEN * DENA * DIETHYLNITROSAMINE * DIETHYLNITROSOAMINE * N,N-DIMETHYL-NITROSAMINE * ETHYLAMINE, N-NITROSODI- * N-NITROSO-ETHANAMINE * NDEA * NITROSO-DIETHYLAMINE * N-NITROSO-DIAETHYLAMINE
N-Nitrosodi-methylamine	DIMETHYLNITROSAMINE * N,N-DIMETHYL-NITROSAMINE * DIMETHYLNITROSOAMINE * DMN * DMNA * N-METHYL-N-NITROSOMETH-ANAMINE * NDMA
N-Nitrosodi-N-butylamine	N-BUTYL-N-NITROSO-1-BUTAMINE * DBN * DBNA * DIBUTYLAMINE, N-NITROSO- * DI-BUTYLNITROSAMINE * DI-n-BUTYLNITRO-SAMINE * N,N-DI-n-BUTYLNITROAMINE * N,N-DIBUTYLNITROSOAMINE * NDBA * N-NITRO-SODIBUTYLAMINE * N-NITROSO-DI-n-BUTYL-AMINE
N-Nitrosodi-propylamine	DI-n-PROPYLNITROSAMINE * DPN * DPNA * NDPA * N-NITROSO-N-PROPYL-1-PROPANA-MINE
N-Nitrosomethyl-ethylamine	ETHYLMETHYLNITROSAMINE * N,N-METHYL-ETHYLNITROSAMINE * N-METHYL-N-NITROSO-ETHAMINE * N-METHYL-N-NITROSOETHYL-AMINE * NEMA * NMEA * N-NITROSOETHYL-METHYLAMINE
N-Nitrosomethyl-vinylamine	ETHENYLAMINE, N-METHYL-N-NITROSO- * N-METHYL-N-NITROSO-ETHENYLAMINE * N-METHYL-N-NITROSOVINYLAMINE * METHYL-VINYLNITROSAMINE * MVNA * NMVA
N-Nitroso-N-Ethylurea	ENU * ETHYLNITROSOUREA * N-ETHYLNITRO-SOUREA * N-ETHYL-N-NITROSO-UREA * 1-ETHYL-1-NITROSOUREA * NEU * NITROSO-ETHYLUREA * NSC 45403

SUBSTANCE	SYNONYMS
N-Nitroso-N-Methylurea	METHYLNITROSOUREA * N-METHYL-N-NITRO-SOUREA * 1-METHYL-1-NITROSOUREA * MNU * N-NITROSO-N-METHYLCARBAMIDE * N-NITRO-N-NITROSOMETHYLUREA * NMH * NMU * NSC 23909 * UREA, 1-METHYL-1-NITROSO-
N-Nitroso-N-methylurethane	ETHYL ESTER of METHYLNITROSO-CARBAMIC ACID * N-METHYL-N-NITROSOCARBAMIC ACID, ETHYL ESTER * N-METHYL-N-NITROSOETHYL-CARBAMATE * METHYLNITROSOURETHANE * N-METHYL-N-NITROSO-URETHANE * MNU * MNUN * NITROSOMETHYLURETHANE * NMUT
N-Nitroso-morpholine	4-NITROSOMORPHOLINE * NMOR
N-Nitroso-nornicotine	NICOTINE, 1'-NITROSO-1'-DEMETHYL- * 1'-DEMETHYNLCOTINE * 1-NITROSO-2-(3-PYRIDYL)-PYRROLIDINE * 3-(1-NITROSO-2-PYRROLIDINYL)-PYRIDINE * NNN
N-Nitrosopiperidine	1-NITROSOPIPERIDINE * NO-Pip * NPIP
N-Nitrosopyrrolidine	NO-Pyr * NPYR
N-Nitrososarcosine	N-METHYL-N-NITROGLYCINE
Oxymetholone	ADRODIN * ADROYD * ANADROL * ANA-DROYD * ANADROSTANO(2,3-c)(1,2,5)OXADI-AZOL-17-OL, 17-METHYL-, (5-alpha,17-beta)-(9CI) * ANAPOLON * ANASTERON * ANA-STERONAL * ANASTERONE * ANADROSTAN-3-ONE, 17-HYDROXY-2-(HYDROXYMETHLENE)-17-METHYL-, (5-alpha,17-beta)- (9CI) 5-alpha-AN-DROSTAN-3-ONE, 17-beta-HYDROXY-2-(HY-DROXYMETHYLENE7-17-METHYL- (8CI) * BECOREL * CI-406 * 4,5-DIHYDRO-2-HYDROXY-METHYLENE-17-alpha-METHYLTESTOSTERONE * DYNASTEN * HMD * 17-HYDROXY-2-(HYDROXY-METHYLENE)-17-METHYL-5-alpha-17-beta-ANDROST-3-ONE * 17-beta-HYDROXY-2-HY-DROXYMETHYLENE-17-alpha-METHYL-3-ANDROSTANONE * 17-beta-HYDROXY-2-(HY-DROXYMETHYLENE)-17-alpha-METHYL-5-alpha-ANDROSTAN-3-ONE * 17-beta-HYDROXY-2-(HYDROXYMETHYLENE)-17-METHYL-5-alpha-ANDROSTAN-3-ONE * 2-HYDROXYMETHYLENE-17-alpha-METHYL-5-alpha-ANDROSTAN-17-beta-OL-3-ONE * 2-HYDROXYMETHYLENE-17-alpha-METHYL-DIHYDROTESTERONE * 2-(HYDROXY-METHYLENE)-71-METHYL DIHYDROTESTERONE * 2-(HYDROXYMETHYLENE)-17-alpha-METHYL-DIHYDROTESTERONE * 2-HYDROXYMETHYLENE-17-alpha-METHYL-17-beta-HYDROXY-3-ANDRO-STANONE * METHABOL * 17-alpha-METHYL-2-HYDROXYMETHYLENE-17-HYROXY-5-alpha-ANDROSTAN-3-ONE * NASTENON * NSC-26,198 * OXIMETHOLONUM * OXIMETOLONA * OXI-

SUBSTANCE	SYNONYMS
Oxymetholone (Cont'd)	TOSONA-50 * OXYMETHENOLONE * PAVISOID * PLENASTRIL * PROTANABOL * ROBORAL * SYNASTERON * ZENALOSYN
Pentachloronitrobenzene	AVICOL * BATRILEX * BOTRILEX * BRASSICOL * EARTHCIDE * FARTOX * FOLOSAN * FOMAC 2 * FUNGICLOR * GC 3944-3-4 * KOBU * KOBUTOL * KP 2 * NCI-C00419 * OLPISAN * PCNB * PENTAGEN * PKhNB * QUINTOCENE * QUINTOZENE * SANICLOR 30 * TERRACHLOR * TERRACLOR * TERRAFUN * TILCAREX * TRI-PCNB * TRITISAN
Phenacetin	p-ACETOPHENETIDE * ACETOPHENETIDIN * ACETOPHENETIDINE * p-ACETOPHENETIDINE * ACETO-4-PHENETIDINE * ACETOPHENETIN * ACET-p-PHENALIDE * p-ACETPHENETIDIN * ACET-p-PHENETIDIN * N-ACETYL-p-PHENETIDINE * p-ETHOXYACETANILIDE * 4-ETHOXYACETANILIDE * 4'-ETHOXYACETANILIDE * N-(4-ETHOXYPHENYL)ACETAMIDE * FENIDINA * FENIA * KALMIN * PERTONAL * PHENACET * PHENACETINE * PHENACITIN * PHENAZETIN * PHENEDINA * PHENIDIN * PHENIN * PYRAPHEN
Phenazopyridine Hydrochloride	2,6-DIAMINO-3-PHENYLAZOPYRIDINE * DIRIDONE * DPP * GASTRACID * MALLOPHENE * NC 150 * AP * PHENAZODINE * 3-(PHENYLAZO)-2,6-PYRIDINEDIAMINE * PHENLYAZO TABLET * PHENAZOPYRIDINE * PIRID * PYRA-
Phenazopyridine Hydrochloride (Cont'd)	ZOFEN * PYRIDACIL * PYRIDIUM * PYRIPYRIDIUM * SEDURAL * URIDINAL * URODINE * W 1655
Phenytoin	ALEVIATIN * ANTISACER * AURANILE * CAUSOIN * CITRULLAMON * CITRULLIAMON * COMITAL * COMITOINA * CONVUL * DANTEN * DANTINAL * DANTOINAL * DANTOINAL KLINOS * DANTOINE * DENYL * DIDAN-TDC-250 * DIFENIN * DIFETOIN * DIFHYDAN * DIHYCON * DI-HYDAN * DIHYDANTOIN * DILABID * DI-LAN * DILANTINE * DILANTIN * DILLANTIN * DINTOIN * DINTOINA * DIAPHANTOIN * DIPHEDAL * DIPHEDAN * DIPHENIN * DIPHENINE * DIPHENTOIN * DIPHENTYN * DIPHENYLAN * DIPHENYLHYDANTOIN * 5,5-DIPHENLYHYDANTOIN * DIPHENYLHYDATANOIN * 5,5-DIPHENYLIMIDAZOLIDIN-2,4-DIONE * 5,5-DIPHENYL-2,4-IMIDAZOLIDINEDIONE * DI-PHETINE * DITOINATE * DPH * EKKO CAPSULES * ELEPSINDON * ENKELFEL * EPAMIN * EPANUTIN * EPASMIR '5' * EPDANTOINE SIMPLE * EPELIN * EPIFENYL * EPIHYDAN * EPILAN * EPILAN-D * EPILANTIN * EPINAT * EPISED * EPTAL * EPTOIN * FENANTOIN * FENIDANTOIN 'S' * FENITOINA * FENTOIN * FENYLEPSIN * FENYTOINE * GEROT-EPILAN-D * HIDAN * HIDANTAL * HIDANTILO * HIDANTINA * HIDANTINA SENOSIAN * HIDANTINA VITORIA * HIDANTOMIN * HINDATAL * HYDANTAL * HYDANTIN * HYDANTOIN * HYDANTOINAL * ICTALIS SIMPLE * IDANTOIL * IDANTOIN *

SUBSTANCE	SYNONYMS
Phenytoin (Cont'd)	KESSODANTEN * LABOPAL * LEPITOIN * LEPSIN * MINETOIN * NCI-C55765 * NEOSHIDANTOINA * NEOSIDANTOINA * NOVANTOINA * OM HIDANTOINA SIMPLE * OMHYDANTOINE * OXYLAN * PHANANTIN * PHANATINE * PHENATINE * PHENATOINE * PHENTOIN * RITMENAL * SACERIL * SANEPIL * SILANTIN * SODANTOIN * SODANTON * SOLANTIN * SOLANTOIN * SOLANTYL * SYLANTOIC * TACOSAL * THILOPHENYL * TOIN * TOIN UNICELLES * ZENTRONAL * ZENTROPIL
Polychlorinated Biphenyls	AROCLOR * AROCLOR 1221 * AROCLOR 1232 * AROCLOR 1242 * AROCLOR 1248 * AROCLOR 1254 * AROCLOR 1260 * AROCLOR 1262 * AROCLOR 1268 * AROCLOR 2565 * AROCLOR 4465 * BIPHENYL, POLYCHLORO- * CHLOPHEN * CHLORINATED BIPHENYL * CHLORINATED DIPHENYL * CHLORINATED DIPHENYLENE * CHLOREXTOL * CHLORO BIPHENYL * CHLORO 1,1-BIPHENYL * CLOPHEN * DYKANOL * FENCLOR * INERTEEN * KANECHLOR * KANECHLOR 300 * KANECHLOR 400 * KANECHLOR 500 * MONTAR * NOFLAMOL * PCB * PCBs * PHENOCHLOR * PHENOCLOR * POLYCHLOROBIPHENYL * PYRALENE * PYRANOL * SANTOTHERM * SANTOTHERM FR * SOVOL * THERMINOL FR-1
Procarbazine	IBENZMETHYZINE * 2-(p-ISOPROPYLCARBAMOYLBENZYL)-1-METHYLHYDRAZINE * N-ISOPROPYL-alpha-(2-METHYLHYDRAZINO)-p-TOLU-
Procarbazine (Cont'd)	AMIDE * 4-((2-METHYLHYDRAZINO)METHYL)-N-ISOPROPYLBENZAMIDE * MATULANE * 1-METHYL 2-(p-(ISOPROPYLCARBAMOYL)BENZYL)-HYDRAZINE * MIH * NATULAN * NSC-77213 * PCB * RO 4-6467
Procarbazine Hydrochloride	IBENZMETHYZIN HYDROCHLORIDE * IBENZMETHYZINE HYDROCHLORIDE * IBZ * 1-(p-ISOPROPYLCARBAMOYLBENZYL)-2-METHYLHYDRAZINE HYDROCHLORIDE * 2-(p-(ISOPROPYLCARBAMOYL)BENZYL)-1-METHYLHYDRAZINE HYDROCHLORIDE * N-ISOPROPYL-alpha-(2-METHYLHYDRAZINO)-p-TOLUAMIDE HYDROCHLORIDE * N-ISOPROPYL-p-(2-METHYLHYDRAZINOMETHYL)BENZAMIDE HYDROCHLORIDE * MBH * p-(N'-METHYLHYDRAZINOMETHYL)-N-ISOPROPYLBENZAMIDE HYDROCHLORIDE * 1-METHYL-2-p-(ISOPROPYLCARBAMYOL)BENZOHYDRAZINE HYDROCHLORIDE * 1-METHYL-2-(p-ISOPROPYLCARBAMOYLBENZYL)HYDRAZINE HYDROCHLORIDE * MIH * NATHULANE * NATULAN * NATULAN HYDROCHLORIDE * NCI-C01810 * NSC-77213 * RO 4-6467
Pronamide	N-(1,1-DIMETHYLPROPYNYL)-3,5-DICHLOROBENZAMIDE * KERB * KERB 50W * PROMAMIDE * PROPYZAMIDE * RH 315
1,3-Propane Sultone	3-HYDROXY-1-PROPANESULPHONIC ACID SULFONE * 1-PROPANESULFONIC ACID-3-HYDROXY-gamma-SULTONE * PROPANESULTONE

SUBSTANCE	SYNONYMS
Beta-Propiolactone	BETAPRONE • BPL • HYDRACRYLIC ACID,beta, LACTONE • 3-HYDROXYPROPIONIC ACID LACTONE • PROPANOLIDE • PROPIOLACTONE • beta-PROPIONOLACTONE • beta-PROPRO-LACTONE
Propylthiouracil	2,3-DIHYDRO-6-PROPYL-2-THIOXO-4(1H)-PYRI-MIDINONE • 2-MERCAPTO-4-HYDROXY-6-n-PROPYLPYRIMIDINE • 2-MERCAPTO-6-PROPYL-4-PYRIMIDONE • 2-MERCAPTO-6-PROPYLPYRI-MID-4-ONE • PROCASIL • PROPACIL • PRO-PYCIL • 6-PROPYL-2-THIO-2,4(1H,3H)PYRIMI-DINEDIONE • PROPYL-THIORIT • PROPYLTHI-OURACIL • 4-PROPYL-2-THIOURACIL • 6-n-PROPYL-2-THIOURACIL • 6-PROPYL-2-THIOURA-CIL • PROTHYCIL • PROPYL-THYRACIL • PRO-THYRAN • PTU • 2-THIO-4-OXO-6-PROPYL-1,3-PYRIMIDINE • 2-THIO-6-PROPYL-1,3-PYRI-MIDIN-4-ONE • 6-THIO-4-PROPYLURACIL • THYREOSTAT II
Reserpine	ALKARAU • ALSERIN • ANQUIL • APOPLON • APSICAL • ASCOSERPINA • AUSTRAPINE • BANASIL • BENAZYL • BIOSERPINE • CARDI-TIVO • CRYSTOSEPINE • DESERPINE • EBERPINE • EBERSPINE • ELSERPINE • ENIPRESSER • ESCAS-PERE • ESERPINE • ESKASERP • GAMMASERPINE • H 520 • HELFOSERPIN • HIPOSERPIL • HISERPIA • IDSOSERP • INTERPINA • KEY-SERPINE • KITINE • 'L,' CARPSERP • LEMISERP • LOWESERP • MAVISERPIN • MAYSERPINE • MEPHASERPIN •
Reserpine (Cont'd)	METHYLRESERPATE 3,4,5-TRIMETHOXYBENZOIC ACID • METHYL RESERPATE 3,4,5-TRIMETHOXY-BENZOIC ACID ESTER • NCI-C50157 • NEO-ANTITENSOL • NEOSERFIN • NEO-SERP • PUR-SERPINE • QUIESCIN • RAUCAP • RAUDIFORD • RAUDIXOID • RAUGAL • RAULEN • RAU-LOYDIN • RAUMORINE • RAUNERVIL • RAUNO RINE • RAUNOVA • RAUPASIL • RAUPOID • RAUSEDAN • RAURINE • RAUSAN • RAU-SED • RAUSEDIL • RAUSEDYL • RAUSERPIN-ALK • RAU-SERPINE • RAUSERPOL • RAUSINGLE • RAUTRIN • RAUVLID • RAUWILID • RAUWIPUR • RAU-WOLEAF • RAWILID • R-E-S • RESEDIN • RESE-DREX • RESERBAL • RESERCAPS • RESERCEN • RESERJEN • RESERLOR • RESERP • RESERPAL • RESERPAMED • RESERPANCA • RESERPENE • RESERPEX • RESERPIL • RESERPINA • RESERPI-NUM • RESERPKA • RESERPOID • RESERPUR • RESIATRIC • RESIDINE • RESINE • RESOCALM • RESPERIN • RESPERINE • RESPITAL • RESTRAN • REZERPIN • RISERPA • RIVASED • RIVASIN • ROLSERP • ROXEL • ROXINOID • ROXYNOID • RYSER • SANDRIL • SANDRON • SARPAGAN • SEDARAUPIN • SEDARAUPINA • SEDERAUPIN • SERFIN • SERFOLIA • SEROLFIA • SERP-AFD • SERPALAN • SERPALOID • SERPANEURONA • SERPANRAY • SERPASIL • SERPASOL • SERPATE • SERPAZIL • SERPAZOL • SERPEDIN • SERPEN • SERPENA • SERPENTIL • SERPENTIN • SERPEN-TINA • SERPICON • SERPIL • SERPILOID • SER-PINE • SERPIPUR • SERPIVATE • SERPIVITE • SERPOGEN • SERPOID • SERPONE • SERPYRIT •

SUBSTANCE	SYNONYMS
Reserpine (Cont'd)	SERTABS • SERTENS • SERTENSIN • SERTINA • TEFASERPINA • TEMPO-RESERPINA • TEMPO-SERPINE • TENSERPINIE • TEPSERPINE • T-SERP • 3,4,5-TRIMETHOXYBENZOYL METHYL RESERPATE • UNILORD • USAF CB-27 • VIO-SERPINE
Saccharin	ANHYDRO-6-SULFAMINEBENZOIC ACID • SSUGRIN VOLLSUSS • 3-BENZISOTHIAZOLI-NONE 1,1-DIOXIDE • 1,2-BENZISOTHIAZOL-3(2H)-ONE 1,1-DIOXIDE • o-BENZOIC SULFI-MIDE • BENZOIC SULPHIMIDE • BENZOIC SULPHIMIDE • o-BENZOIC SULPHIMIDE • o-BENZOSULFIMIDE • o-BENZOSULPHIMIDE • BENZOSULPHIMIDE • BENZO-2-SULPHIMIDE • BENZO-SULPHINIDE • o-BENZOYL SULFIMIDE • o-BENZOYL SULPHIMIDE • 1,2-DIHYDRO-2-KETOBENZISOSULFONAZOLE • 1,2-DIHYDRO-2-KETOBENZISOSULPHONAZOLE • 2,3-DIHYDRO-3-OXOBENZISOSULFONAZOLE • 2,3-DIHYDRO-3-OXOBENZISOSULPHONAZOLE • GARANTOSE • GLUCID • GLUSIDE • HERMESETAS • 3-HYDROXYBENZISOTHIAZOLE-S,S-DIOXIDE • NATREEN • INSOLUBLE SACCHARIN • KANDISET • SACARINA • SACCHARIMIDE • SACCHARINA • SACCHARIN ACID • SACCHARINE • SAC-CHARIN INSOLUBLE • SACCHARINOL • SAC-CHARINOSE • SACCHAROL • SAXIN • SUCRE EDULCOR • SUCRETTE • o-SULFOBENZIMIDE • o-SULFOBENZOIC ACID IMIDE • 2-SULPHO-BENZOIC IMIDE • SYKOSE • ZAHARINA
Safrole	5-ALLYL-1,3-BENZODIOXOLE • ALLYLCATECHOL METHYLENE ETHER • ALLYLDIOXYBENZENE METHYLENE ETHER • 1-ALLYL-3,4-METHYLENE-DIOXYBENZENE • 4-ALLYL-1,2-METHYLENEDI-OXYBENZENE • m-ALLYLPYROCATECHIN METH-YLENE ETHER • 4-ALLYLPYROCATECHOL FOR-MALDEHYDE ACETAL • ALLYLPYROCATECHOL METHYLENE ETHER • 1,3-BENZODIOXOLE, 5-ALLYL • 1,2-METHYLENEDIOXY-4-ALLYLBENZENE • 3,4-METHYLENEDIOXY-ALLYBENZENE • 5-(2-PROPENYL)-1,3-BENZODIOXOLE • RHYUNO OIL • SAFROL • SAFROLE MF • SHIKIMOLE • SHIKOMOL
Selenium Sulfide	NCI-C50033 • SELENIUM MONOSULFIDE • SELENIUM SULPHIDE
Soots, Tars & Mineral Oils	NONE
Streptozotocin	2-DEOXY-2-(((METHYLNITROSOAMINO)CAR-BONYL)AMINO)-D-GLUCOPYRANOSE • 2-DEOXY-2-(3-METHYL-3-NITROSOUREIDO)-D-GLUCOPYRANOSE • N-d-GLUCOSYL(2)-N'- • N-D-GLUCOSYL-(2)-N'-NITROSOMETHYLUREA • NCI-C03167 • NSC-85998 • STR • STREPTO-ZOTICIN • U-9889
2,3,7,8-Tetrachloro-dibenzo-p-dioxin	DIBENZO(b,e)(1,4)DIOXIN, 2,3,7,8-TETRACHLO-RO- • DIOXIN (HERBICIDE CONTAMINANT) • NCI-C03714 • TCDBD • TCDD • 2,3,7,8-TCDD • 2,3,7,8-TETRACHLORODIBENZO(b,e)(1,4)DIOXAN • 2,3,7,8-TETRACHLORODIBENZO-1,4-DIOXIN

SUBSTANCE	SYNONYMS
Tetrachloroethylene	ANKILOSTIN * ANTISOL 1 * CARBON BICHLORIDE * CARBON DICHLORIDE * DEE-SOLV * DIDAKENE * DOW-PER * ENT 1,860 * ETHYLENE TETRACHLORIDE * FEDAL-UN * NEMA * NCI-C04580 * PER * PERAWIN * PERC * PERCHLOR * PERCHLORETHYLENE * PERCHLOROETHYLENE * PERCLENE * PERCOSOLVE * PERK * PERKLONE * PERSEC * TETLEN * TETRACAP * TETRACHLORETHYLENE * TETRACHLOROETHENE * TETRACHLOROETHYLENE (DOT) * 1,1,2,2-TETRACHLOROETHYLENE * TETRALEX * TETRALENO * TETRAVEC * TETROGUER * TETROPIL
Thioacetamide	ACETOTHIOAMIDE * ETHANETHIOAMIDE * TAA * THIACETAMIDE * USAF CB-21 * USAF EK-1719
Thiourea	SULOUREA * THIOCARBAMIDE * 2-THIOUREA * THU * SUAF EK-497
Thorium Dioxide	THORIA * THOROTRAST * THORTRAST * UMBRATHOR
o-Toluidine Hydrochloride	1-AMINO-2-METHYLBENZENE HYDROCHLORIDE * 2-AMINO-1-METHYLBENZENE HYDROCHLORIDE * 2-AMINOTOLUENE HYDROCHLORIDE * o-AMINOTOLUENE HYDROCHLORIDE * 1-METHYL-2-AMINOBENZENE HYDROCHLORIDE * 2-METHYL-1-AMINOBENZENE HYDROCHLORIDE * o-METHYLANILINE HYDROCHLORIDE * 2-METHYLANILINE HYDROCHLORIDE * o-METHYL-
o-Toluidine Hydrochloride (Cont'd)	BENZENAMINE HYDROCHLORIDE * 2-METHYLBENZENAMINE HYDROCHLORIDE * NCI-C02335 * 2-TOLUIDINE HYDROCHLORIDE * o-TOLYLAMINE HYDROCHLORIDE
Toxaphene	AGRICIDE MAGGOT KILLER (F) * ALLTEX * ALLTOX * CAMPHECHLOR * CAMPHOCHLOR * CAMPHOCLOR * CAMPHOFENE HUILEUX * CHEM-PHENE * CHLORINATED CAMPHENE * CHLOROCAMPHENE * CLOR CHEM T-590 * COMPOUND 3956 * CRESTOXO * CRISTOXO * CRISTOXO 90 * ENT 9,735 * ESTONOX * FASCOTERPENE * GENIPHENE * GY-PHENE * HERCULES 3956 * HERCULES TOXAPHENE * KAMFOCHLOR * M 5055 * MELIPAX * MOTOX * NCI-C00259 * OCTACHLOROCAMPHENE * PENPHENE * PHENACIDE * PHENATOX * POLYCHLORCAMPHENE * POLYCHLORINATED CAMPHENES * POLYCHLOROCAMPHENE * STROBANE-T * SYNTHETIC 3596 * TOXADUST * TOXAKIL * TOXAPHENE (DOT) * TOXON 63 * TOXYPHEN * VERTAC 90%
Trichlorethylene	ACETYLENE TRICHLORIDE * ALGYLEN * ANAMENTH * BENZINOL * BLACOSOLV * BLANCOSOLV * CECOLENT * CHLORIDEN * 1-CHLORO-2,2-DICHLOROETHYLENE * CHLORYLEA * CHLORYLEN * CHORYLEN * CIRCOSOLV * CRAWHASPOL * DENSINFLUAT * 1,1-DICHLORO-2-CHLOROETHYLENE * DOW-TRI * DUKERON * ETHINYL TRICHLORIDE * ETHYLENE TRICHLORIDE * FLECK-FLIP * FLOCK FLIP * FLUATE * GEMALGENE * GERMALGENE * LANADIN *

SUBSTANCE	SYNONYMS
Trichlorethylene (Cont'd)	LETHURIN * NARCOGEN * NARKOGEN * NARKOSOID * NCI-C04546 * NIALK * PERM-A-CHLOR * PERM-A-CLOR * PETZINOL * PHILEX * TCE * THRETHYLEN * THRETHYLENE * TRETHYLENE * TRI * TRIAD * TRIAL * TRIASOL * TRICHLORAN TRICHLOREN * TRICHLOROETHENE * TRICHLOROETHYLENE * 1,1,2-TRICHLOROETHYLENE * 1,2,2-TRICHLOROETHYLENE * TRICHLOROETHYLENE (DOT) * TRI-CLENE * TRIELENE * TRIELIN * TRIKLONE * TRILEN * TRILENE * TRILINE * TRIMAR * TRIOL * TRI-PLUS * TRI-PLUS M * VESTROL * VITRAN * WESTROSOL
Tris(1-aziridinyl)-phosphine Sulfide	AZIRIDINE, 1,1,1'-PHOSPHINOTHIOYLIDYNETRIS- * GIROSTAN * NCI-C01649 * NSC-6396 * ONCOTEPA * 1,1',1''-PHOSPHINOTHIOYLIDYNETRISAZIRIDINE * PHOSPHOROTHIOIC TRIAMIDE, N,N',N'-TRIETHYLENE * TESPA * TESPAMIN * TESPAMINE * THIOPHOSPHAMIDE * THIOPHOSPHORAMIDE, N,N',N'-TRIETHYLENE- * THIO-TEP * TRIO-TEPA * THIOTRIETHYLENEPHOSPHORAMIDE * TIFOSYL * TRIAZIRIDINYLPHOSPHINE SULFIDE * N,N',N''-TRI-1,2-ETHANEDIYLPHOSPHOROTHIOIC TRIAMIDE * N,N',N''-TRI-1,2-ETHANEDIYLTHIOPHOSPHORAMINE * TRI(ETHYLENEIMINO)THIOPHOSPHORAMIDE * N,N',N''-TRIETHYLENEPHOSPHOROTHIOIC TRIAMIDE * N,N',N'-TRIETHYLENETHIOPHOSPHAMIDE * N,N',N'-TRIETHYLENETHIOPHOSPHORAMIDE * TRIETHYLENETHIOSPHOROTRIAMIDE * TRIS(1-AZIRIDINYL)PHOSPHINE SULPHIDE * TRIS(ETHYLENIMINO)THIOPHOSPHATE * TSPA
Tris(2,3-dibromopropyl)-phosphate	ANFRAM 3PB * APEX 462-5 * BROMKAL P 67-6HP * 2,3-DIBROMO-1-PROPANOL PHOSPHATE * (2,3-DIBROMOPROPYL) PHOSPHATE * ES685 * FIREMASTER LV T 23P * FIREMASTER T23P * FIREMASTER T23P-LV * FLACAVON R * FLAMEX T 23P * FLAMMEX AP * FLAMMEX T 23P * NCI-C03270 * T23P * TRIS * TRIS-BP * TRIS(DIBROMOPROPYL)PHOSPHATE * TRIS(2,3-DIBROMOPROPYL) PHOSPHORIC ACID ESTER * USAF DO-41 * ZETIFEX ZN
Trypan Blue, commercial grade	AMANIL SKY BLUE * AMANIL SKY BLUE R * AMADINE BLUE 4B * AZIDINBLAU 3B * AZIDINE BLUE 3B * AZURRO DIRETTO 3B * BENCIDAL BLUE 3B * BENZAMINBLAU 3B * BENZAMINE BLUE * BENZAMINE BLUE 3B * BENZANIL BLUE 3BN * BENZANIL BLUE R * BENZOBLAU 3B * BENZO BLUE * BENZO BLUE 3B * BENZO BLUE 3BS * BLEU DIAMINE * BLEU DIAZOLE N 3B * BLEU DIRECTE 3B * BLEUE DIRETTO 3B * BLEU TRYPANE N * BLUE 3B * BLUE EMB * BRASILAMINA BLUE 3B * BRASILAZINA BLUE 3B * CENTRALINE BLUE 3B * CHLORAMIBLAU 3B * CHLORAMINE BLUE * CHLORAMINE BLUE 3B * CHLORAZOL BLUE 3B * CHROME LEATHER BLUE 3B * C.I. 23850 * C.I. DIRECT BLUE 14 * C.I. DIRECT BLUE 14, TETRASODIUM SALT * CONGOBLAU 3B * CONGO BLUE * CONGO BLUE 3B * CRESOTIN BLUE 3B * DIAMINBLAU 3B * DIAMINEBLUE * DIAMINE BLUE 3B * DIANILBLAU * DIANILBLAU H3G * DIANIL BLUE * DIANIL BLUE H3G * DIAPHTAMINE BLUE TH * DIAZINE BLUE

SUBSTANCE	SYNONYMS
ypan Blue, ommercial grade (Cont'd)	3B • DIAZOL L 3B • DIPHENYL BLUE 3B • DIRECTBLAU 3B • DIRECT BLUE 3B • DIRECT BLUE 3BX • DIRECT BLUE D3B • DIRECT BLUE FFN • DIRECT BLUE H3G • DIRECT BLUE M3B • DIRECTAKOL BLUE 3BL • HISPAMIN BLUE 3BX • NAPHTAMINE BLUE 2B • NAPHTAMINE BLUE 3BX • NAPHTHAMINBLAU 3BX • NAPHTHAMINE BLUE 3BX • NAPHTHYLAMINE BLUE • NIAGARA BLUE • NIAGARA BLUE 3B • ORION BLUE 3B • PARAMINE BLUE 3B • PARKIBLEU • PARKIPAN • PONTAMINE BLUE 3BX • PYRAZOL BLUE 3B • PYROTROPBLAU • RENOLBLAU 3B • SODIUM DITOLYDISAZOBIS-8-AMINO-1-NAPHTHOL-3,6-DISULFONATE • SODIUM DITOLYDISAZO-BIS-8-AMINO-1-NAPHTHOL-3,6-DISULPHONATE • TRIANOL DIRECT BLUE 3B • TRIAZOLBLAU 3BX • TRIPAN BLUE • TRYPAN BLUE BPC • TRYPAN BLUE SODIUM SALT • TRYPANE BLUE
Uracil Mustard	AMINOURACIL MUSTARD • 5-(BIS(2-CHLORO-ETHYL)AMINO)-2,4(1H,3H)PYRIMIDINEDIONE • 5-(BIS(2-CHLOROETHYL)AMINO)URACIL • 5,N,N-BIS(2-CHLOROETHYL)AMINOURACIL • CB-4835 • DEMETHYLDOPAN • DESMETHYLDOPAN • 5-(DI-(beta CHLOROETHYL)AMINO)URACIL • 5-(DI-2-CHLOROETHYL)AMINOURACIL • 2,6-DIHYDROXY-5-BIS(2-CHLOROETHYL)AMINO-PYRIMIDINE • ENT 50439 • NCI-C04820 • NSC-34462 • CHLORETHAMINACIL • NORDOPAN • SK 19849 • U-8344 • URAMUSTINE

SUBSTANCE	SYNONYMS
Urethane	ESTANE 5703 • ETHYL CARBAM... ETHYLURE THAN • ETHYL URETHANE • ETHYL URETHANE O-ETHYLURETHANE • LEUCETHANE • LEUCO-THANE • NSC 746 • PRACARBAMINE • PRACA BAMIN • URETHAN
Vinyl Chloride	CHLORETHENE • CHLORETHYLENE • CHLORO ETHENE • CHLOROETHYLENE • ETHYLENE MON OCHLORIDE • MONOCHLOROETHENE • MON CHLOROETHYLENE • MONOCHLOROETHYLEN (DOT) • TROVIDUR • VC • VCM • VINYL CHL RIDE (DOT) • VINYL CHLORIDE MONOMER • VINYL C MONOMER
Vinylidene Chloride	1,1-DICHLOROETHENE (9CI) • 1,1-DCE • 1,1-DICHLOROETHYLENE • ETHENE, 1,1-DICHLOR • NCI-C54262 • SCONATEX • VDC • VINYLI-DENE CHLORIDE (II) • VINYLIDENE DICHLORI • VINYLILINE CHLORIDE

APPENDIX II

Assembly Bill No. 3011

Passed the Assembly August 26, 1982

/s/ James D. Driscoll

Chief Clerk of the Assembly

Passed the Senate August 24, 1982

/s/ Darryl R. White

Secretary of the Senate

This bill was received by the Governor this 30th day of August, 1982, at 2 o'clock p.m.

/s/ Esther Irving

Private Secretary of the Governor

Attachment #1-44

CHAPTER ———

An act to add and repeal Section 3212.1 of the Labor Code, relating to workers' compensation.

LEGISLATIVE COUNSEL'S DIGEST

AB 3011, Torres. Workers' compensation: firefighters.

Existing law provides that in the case of specified law enforcement and firefighting employees, an injury under the workers' compensation law includes hernia, heart trouble, and pneumonia which develops or manifests itself during the period of the specified employment.

This bill would, in addition, provide that in the case of active volunteer or paid firefighting members of state and local fire departments, an injury under the workers' compensation law includes cancer which develops or manifests itself during the period of the specified employment, provided the member demonstrates exposure during the employment to a known carcinogen, as defined, which is reasonably linked to the disabling cancer.

This bill would also provide that the cancer so developing or manifesting itself is rebuttably presumed to arise out of and in the course of the employment.

Article XIII B of the California Constitution and Sections 2231 and 2234 of the Revenue and Taxation Code require the state to reimburse local agencies and school districts for certain costs mandated by the state. Other provisions require the Department of Finance to review statutes disclaiming these costs and provide, in certain cases, for making claims to the State Board of Control for reimbursement.

This bill would provide that no appropriation is made by this act for the purpose of making reimbursement pursuant to the constitutional mandate or Section 2231 or 2234, would recognize that local agencies and school districts may pursue their other available remedies to seek reimbursement for these costs.

This bill would require that all reimbursements to a local agency or school district or any state agency pursuant to the bill be paid from the appropriation to the Department of Industrial Relations for the payment of additional compensation for subsequent injury as provided in specified provisions.

This bill, in compliance with Section 2231.5 of the Revenue and Taxation Code, would also repeal, as of January 1, 1989, the provisions contained in the bill for which state reimbursement is required.

The people of the State of California do enact as follows:

SECTION 1. Section 3212.1 is added to the Labor Code, to read:

3212.1. In the case of active firefighting members of fire departments of cities, counties, cities and counties, districts, or other public or municipal corporations or political subdivisions, and active fire fighting members of the fire departments of the University of California, whether these members are volunteers, partly paid, or fully paid, and in the case of active firefighting members of the Department of Forestry, or of any county forestry or firefighting department or unit, whether volunteers, partly paid, or fully paid, the term "injury" as used in this division includes cancer which develops or manifests itself during a period while the member is in the service of the department or unit, provided that the member demonstrates that he or she was exposed, while in the service of the department or unit, to a known carcinogen as defined by the International Agency for Research on Cancer, or as defined by the director, and that the carcinogen is reasonably linked to the disabling cancer.

The compensation which is awarded for cancer shall include full hospital, surgical, medical treatment, disability indemnity, and death benefits, as provided by the provisions of this division.

The cancer so developing or manifesting itself in these cases shall be presumed to arise out of and in the course of the employment. This presumption is disputable and may be controverted by other evidence, but unless so controverted, the appeals board is bound to find in accordance with it. This presumption shall be extended to a member following termination of service for a period of three calendar months for each full year of the requisite service, but not to exceed 60 months in any circumstance, commencing with the last date actually worked in the specified capacity.

This section shall remain in effect only until January 1, 1989, and as of this date is repealed, unless a later enacted statute, which is chaptered before January 1, 1989, deletes or extends this date.

SEC. 2. Notwithstanding Section 6 of Article XIII B of the California Constitution and Section 2231 or 2234 of the Revenue and Taxation Code, no appropriation is made by this act for the purpose of making reimbursement pursuant to these sections. It is recognized, however, that a local agency or school district may pursue any remedies to obtain reimbursement available to it under Chapter 3 (commencing with Section 2201) of Part 4 of Division 1 of that code. However, notwithstanding any other provision f law to the contrary, all reimbursements to a local agency or school district or any state agency pursuant to this act shall be paid from the appropriation to the Department of Industrial Relations for the payment of additional compensation for subsequent injury, as provided in Article 5 (commencing with Section 4750) of Chapter 2 of Part 2 of Division 4 of the Labor Code.

Approved September 30, 1982

/s/ Edmund G. Brown

Governor

APPENDIX III

CANCER INFORMATION SERVICE (CIS)

Cancer Information Service (CIS) offices are affiliated with Comprehensive Cancer Centers. These centers are special research and treatment centers recognized by the National Cancer Institute. CIS offices are staffed by trained persons who can provide support and access to the latest information on cancer and local resources. CIS offices do not diagnose cancer or recommend treatment for individual cases. All calls are kept confidential and you do not have to give your name.

State	*Phone Number*
Alabama	800-292-6201
Alaska	800-638-6070
California (Area Codes 213, 714, 805 only)	800-252-9066
Colorado	800-332-1850
Connecticut	800-922-0824
Delaware	800-523-3586
District of Columbia	202-636-5700
Florida	800-432-5953
Georgia	800-327-7332
Hawaii (Oahu)	808-524-1234
Illinois	800-972-0586
Kentucky	800-432-9321
Maine	800-225-7034
Maryland	800-492-1444
Massachusetts	800-952-7420
Minnesota	800-582-5262
Montana	800-525-0231
New Hampshire	800-225-7034
New Jersey (Northern)	800-223-1000
New Jersey (Southern)	800-523-3586
New Mexico	800-525-0231
New York State	800-462-7255
New York City	212-794-7982
North Carolina	800-672-0943
North Dakota	800-328-5188
Ohio	800-282-6522
Pennsylvania	800-822-3963
South Dakota	800-328-5188
Texas	800-392-2040
Vermont	800-225-7034
Washington	800-552-7212
Wisconsin	800-362-8038
Wyoming	800-525-0231
For All Other States	800-638-6694

Attachment #1-47

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pational Cancer—Prevention and Control
upational Safety and Health Series No. 39
International Labour Office
CH-1211 Geneva 22, Switzerland

Occupational Diseases: A Guide to Their Recognition
National Institute for Occupational Safety and Health
Center for Disease Control
Public Health Service
U.S. Department of Health and Human Services
DHHS (NIOSH) Publication No. 77-181

Occupational Mortality Among Professional Firefighters
James Lloyd Abrams
University of Oklahoma Graduate College
Oklahoma City, Oklahoma

Second Annual Report on Carcinogens
National Toxicology Program
Public Health Service
U.S. Department of Health and Human Services
NTP 81-43

Science and Cancer
National Cancer Institute
National Institutes of Health
Public Health Service
U.S. Department of Health and Human Services
NIH Publication No. 80-568

Support Document Test Data Development Standards: Chronic Health Effects
Health Review Division
Office of Toxic Substances
Environmental Protection Agency
EPA 560/11-79-001

What You Should Know About Cancer
National Cancer Institute
National Institutes of Health
Public Health Service
U.S. Department of Health and Human Services
NIH Publication No. 79-1828

Attachment #1-49

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I am speaking in favor of House Bill 2961

I am a widow of a Kansas City, Kansas police officer.

My husband, Carl E. Winterringer, served on the Kansas City, Kansas Police Department from April 1, 1956 until April 10, 1990. Beginning as a patrolman and retiring as Commander of the Detective Bureau.

During these years, I also worked to supplement our income, while raising three children, mostly on my own as he spent many additional hours working without pay serving the police department and the residents of Kansas City, Kansas.

He was one of the first members of the Metro Squad when it was formed and it was not unusual for him to work 16 to 18 hours a day, 7 days a week, NO OVERTIME PAY.

I could go into a lot of stories and detail about what it is like being a police officers wife, however, you yourself put in many extra hours serving the public and the people of this State while your families are at home living the same way that our family did.

The State laws use to require that a person be married to a police officer when he retired or they were not eligible for his pension upon his death. I have to say that I agreed with that law. However, it has been changed and if a person marries a retired police officer and they are married five years before he passes away, that person now receives his pension. My question is what did she do to deserve it?

Carl and I were married for 36 years and one week, before he passed away from cancer. He always said, "Don't give up my pension, you earned it right along with me and if you meet a nice man, you'll just have to shack up".

However, neither Carl or I were raised that way. He died in 1987 at the age of 57. I am still not an old lady and I have met a nice man.

I am not alone in this situation. There are many widows of police officers and firefighters that are in the same situation as I am. If we marry we lose the pensions that we also worked for.

Consider the fact that some of these widows are much younger, with children, than myself. My son is also a Kansas City, Kansas police officer and has his time vested for his pension. He is 36 years old. Many officers are in this situation and if something happens to them, you are requiring that their wives never marry, lose the pension or live in sin.

It is time that you give us our rights and our dignity back. You have given the pension to women who did not earn it, but ignored the ones who did.

When you are considering this House Bill, also consider your spouse and children's future. I am only familiar with laws that concern police officers and fire fighters families, but realize that many other persons on KPERS could be in the same situation.

Patricia M. Winterringer
474 N. Neconi
Bonner Springs, Kansas 66012

Pensions, Investments & Benefits
Attachment #2
3-4-92

HOUSE OF REPRESENTATIVES
STATE OF KANSAS

STATE CAPITOL, ROOM 330-N
TOPEKA, KANSAS 66612-1591
(913) 296-7643

REPRESENTATIVE, THIRTY-SEVENTH DISTRICT
WYANDOTTE COUNTY
2206 EVERETT
KANSAS CITY, KANSAS 66102-2602

TOPEKA

WILLIAM J. REARDON
SPEAKER PRO TEM

COMMITTEE ASSIGNMENTS
VICE-CHAIRMAN: EDUCATION
MEMBER: TAXATION
LEGISLATIVE, JUDICIAL AND CONGRESSIONAL APPORTIONMENT
CALENDAR AND PRINTING
LEGISLATIVE COORDINATING COUNCIL

Testimony
of
Speaker Pro Tem Bill Reardon

HB 2970
House Pensions and Investments Committee
March 4, 1992

Thank you, Mr. Chairman and members of the committee. I appreciate your attention to HB 2970 which addresses the issue of the waiting period for KPERS disability benefits. This issue was brought to my attention by Mr. Charles Rentfro, of Kansas City, Kansas, who has experienced the negative impact of the current law.

Mr. Rentfro is with us today and will explain the details of his personal experience. He came to me requesting legislation to correct what I agree is an injustice in the law. To his credit, Mr. Rentfro did not ask for this change for his own personal benefit, but rather for other future KPERS disability beneficiaries.

To me, this is a simple matter of justice. This amendment is necessary to assure that disabled persons are not penalized for attempting, in good faith, to return to work after being disabled from an injury or sickness.

Under current law, there is a 180 day waiting period before an individual is entitled to long-term disability benefits. The problem arises when a person who has been off on disability for a period short of 180 days returns to work. If it becomes apparent that the person is unable to continue working and is sent home, the 180 day waiting period starts over. Thus you could have an individual, who is disabled as a result of sickness

or injury, who has been off of work for 179 days. He truly believes that he is capable of returning to work and attempts to do just that. However it quickly becomes apparent to everyone that the worker is still suffering from the disability and is unable to perform his duties. The worker is sent home the same day that he had returned to work. Under current law, the worker, who would have been entitled to long-term disability benefits had he chosen not to try to return to work, will now have to wait an additional 180 days before receiving benefits which would have been rightfully his.

The Social Security Administration follows a similar plan as I am proposing. Under Social Security regulations, the waiting period for long-term disability is approximately 180 days depending on what day of the month the disability starts. If a worker returns to work but is later unable to continue due to the earlier disability, they are not penalized for the period they are unable to work. The Social Security Administration is concerned only with the "date of onset" of the disability. The waiting period does not start over unless it is a different disability that causes the loss of work.

Under the proposed amendment, the waiting period for long-term benefits would not start over until six months have passed from the time the individual returned to work. After six months, the disability would be deemed the result of a new sickness or injury and the waiting period would start all over again.

The amendment is necessary to protect an individual who, in good faith tries to do the right thing. We should not penalize the individuals that are trying to return to the work force.

I respectfully ask you to pass HB 2970 favorably out of your committee.

STATE OF KANSAS EMPLOYEE ALERT!

YOU SHOULD BE AWARE OF THE FOLLOWING FACTS ABOUT THE CURRENT CONDITION OF YOUR RETIREMENT SYSTEM:

- As an active state employee you are contributing, by law, 4% of you pay toward your retirement benefits while the state is contributing only 3.3%. Your participation and the amount contributed are mandatory!

- In 1987, state employees were merged with USD employees in an effort to reduce the employer KPERS contribution rate. This was done even though salaries for the USD employees are controlled by the school districts and are often higher on average than the salaries for state employees, together with the fact that USD employees did not begin paying into KPERS until nearly ten years after state employees. Given these differences and the fact that the state employees' plan should be nearly funded, why are the employer contribution rates to KPERS the same for state and school employees? Is the employer contribution rate for state employees really being used towards state employees' benefits or is it being applied towards benefits for USD employees? It should be noted that the state has a definite interest in the employer contribution rate for USD employees since payment is made from the STATE GENERAL FUND!

- Employees covered by the Regents retirement system are provided an 8% employer contribution rate to fund their retirement benefits, compared to the 3.3% employer contribution rate provided for the majority of other state employees.

- The employer contribution rate for state employees has continued to drop each year and in fact has been decreased a year in advance by the Legislature and promoted as savings to the taxpayer. In reality, your contribution dollars are the primary source used to fund your retirement which allows state funds formerly dedicated to your retirement to be spent elsewhere. The so-called "savings" is a one-sided perspective which fails to acknowledge the utilization of these "savings" to fund other programs, including the retirement program for other classes of employees. Is this merely another form of TAX placed on state employees?

- Recent studies conducted by other states conclude that Kansas ranks among the lowest in the United States in the amount of the annual retirement benefit paid and is the only state which has an employer contribution rate lower than the employee contribution rate! If the retirement program for state employees is in a financial condition sound enough to lower the employer contribution rate each year, then why are benefits still so poor?

Pensions, Investments & Benefits
Attachment #4
3-4-92

- The original intent at the formation of KPERS was that employee and employer contributions towards retirement benefits would be equally shared in the goal to accumulate adequate funds for payment of these benefits. The cost of employee benefits for years of service prior to the state's membership in KPERS (employees who worked for the state prior to 1962), the cost of group life and disability insurance and the cost of administration of KPERS were to be paid by the employer. The original employer contribution rate was set at 5.35% for this purpose. However, the facts show that plan administrators have either lost sight of these original promises or have altered them, either intentionally or unintentionally, with new practices.

- There currently is a proposal to eliminate the future employee contribution rate to KPERS and have your retirement benefits solely funded by the state. This may appear to be in the best interest of state employees since the amount formerly deducted for KPERS would now be included in take-home pay. It may even be promoted as a pay increase or may be used in lieu of a cost of living increase. On the surface this would appear to be to the advantage of state employees. However, this is not a pay increase and it should not take the place of future pay increases. State employees will not receive any more than they did before. The same amount of <u>your</u> money will simply be available to you for other uses, with no additional salary cost to the state.

- What affect will this proposal to eliminate future employee contributions have on the contributions and earnings you currently have accumulated in the retirement system? The action would remove the appearance of "TRUST FUND" and allow decision makers to gain control of the present $4 BILLION of accumulated assets in the KPERS fund. <u>Does this mean that employees who left state service could not withdraw their own money?</u> When the retirement system becomes fully funded who will profit from the excess, the state through reduced contributions or the employees through improvement of benefits? Will this simply be a method to raid the pension fund by lowering state contributions in order to balance the state budget?

- As a state employee, would you rather have the employer match your contribution and use any additional money to improve your benefits or have your retirement solely funded and controlled by the state? Should the state as an employer provide some level of post-retirement health insurance benefits or benefit increases to offset inflation?

- If your are concerned about these facts, <u>write to your legislator and to the PENSION STUDY COMMITTEE</u> before their next meeting on March 6, 1992 to voice your opinion on these issues.
  Address your letter to:

Representative Don Resac
Room 278-W, State Capitol
Topeka, Kansas 66612

Senator Marge Petty
Room 523-S, State Capitol
Topeka, Kansas 66612

continue its practice of not supporting or opposing Supre[illegible]ourt nominations.

Whe[illegible]mas was EEOC chairman, the a[illegible]ncy "adopted policies and engaged in actions that significantly diminished the rights of older workers" under ADEA, the statement had said.

During Senate Judiciary Committee confirmation hearings on his nomination to the Supreme Court, Thomas disavowed statements in a 1985 interview in which he indicated he believed some forms of age discrimination were justified. "I have never condoned violations of the ADEA," Thomas told Sen. Howard M. Metzenbaum, D-Ohio, who asked about quotations in the ABA Banking Journal.

In the 1985 interview, Thomas said, "I am of the opinion that there are many technical violations of the ADEA that, for practical or economic reasons, make sense. Older workers cost employers more most experienced and costly part of their work[illegible]rce."

Thomas at Senate hearing

Thomas indicated he no longer felt that way. "These efforts on the part of employers may make sense to them," he told the Senate panel. "But if they are wrong, they are wrong. If they violate the act, they violate the act."

While Thomas' EEOC record was brought up at the hearings, much of the questioning dealt with his views on natural law, a legal doctrine based on the thesis that some human rights transcend man-made laws, and on abortion.

Thomas said his advocacy of natural law would not cloud his judgment as a justice, and declined to discuss his views on abortion.

Congress set to probe states' raiding of pensions

Alarmed at the growing number of states that are raiding public pension coffers to balance their budgets, the House Select Committee on Aging starts hearings this month to map the extent of the problem and explore ways to curb the practice.

Nearly 17 million state and local government employees and retirees are enrolled in 2,400 pension systems with assets of about $750 billion. Nearly 20 states have tapped their pension funds in recent years, often by withdrawing money, reducing their contributions or altering the accounting assumptions that determine their contribution levels.

Roybal

State and local governments see these funds "as an easy way to address current budget problems," says committee chairman Edward R. Roybal, D-Calif., in announcing the hearings.

The most visible raid: in California last summer, state lawmakers shifted $1.6 billion from the $63 billion California Public Employees Retirement System to help balance the state budget.

"We're seeing this as the beginning of a trend that could gain momentum," says a committee aide. Raids also have occurred in such bellwether states as New York and Texas.

Public pensions are not protected by the Employee Retirement Income Security Act (ERISA), the federal law that sets standards for private-sector pensions. Public employee unions have tried to secure legislative safeguards for their pensions but generally have been unsuccessful, say committee aides.

Among possible federal safeguards: prudent investment rules, full disclosure requirements, minimum fiduciary standards and prohibitions on using public pension assets for non-retirement purposes.

A big roadblock looms, however. It is unclear whether Congress has the constitutional power to tell states how to run their public employee pension systems. "There is no clear Supreme Court ruling in this area," says a spokesman. "That's one of the issues we'll be confronting."

AARP BULLETIN

October 1991
Volume XXXII, Number 9

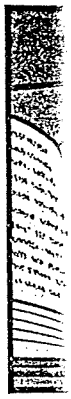
AARP Bulletin (ISSN 1044-1123) is published monthly except August by the American Association of Retired Persons, 3200 E. Carson St., Lakewood, Calif. 90712. Annual membership dues are $5, which includes 85 cents for annual subscription to the *AARP Bulletin.*

Second-class postage paid at Lakewood, Calif., and at additional mailing offices. POSTMASTER: Send address changes to *AARP Bulletin*, c/o AARP, P.O. Box 199, Long Beach, Calif. 90801. Editorial offices at 601 E St. N.W., Washington, D.C. 20049 (telephone [202] 434-2277).

[illegible]ence Thomas attra[illegible] positive re[illegible]onses. of those who w[illegible] calle[illegible] derstood *why* A[illegible] expre[illegible] Thomas' record while chair[illegible] ment Opportunity Commis[illegible]

It is really quite simple. E[illegible] Dr. Ethel Percy Andrus, fi[illegible] discrimination in employm[illegible] ago, we have been an advo[illegible] ing number of older worke[illegible] belong to our Association. future members have a stak[illegible] in the work force.

It is for that reason—*an[illegible]* although AARP did not su[illegible] Thomas' nomination to the felt compelled to raise qu[illegible] as EEOC chairman in the government's chief enforc[illegible] tion in Employment Act (A[illegible] in an examination of his qualifications to be a Supreme Court justice.



The main points we focused on:

- EEOC's refusal to implement rules requiring employers to make pension contributions for workers over age 65 cost older workers *$450 million per year* in lost benefits until Congres[illegible]
- An EEOC decision tha[illegible] programs are not covere[illegible] effect of *excluding* older w[illegible] programs.
- More than 13,000 f[illegible] charges by older workers [illegible] because the agency failed[illegible] the required two-year per[illegible]
- EEOC issued a rule t[illegible] force older workers to g[illegible] the ADEA in order to r[illegible] benefits. (Congress ultima[illegible]

AARP *should* be concer[illegible] Thomas' beliefs on such personal politics, are *not* in[illegible] zation. AARP has *never* s[illegible] candidate or nominee for a[illegible] of his or her political part[illegible] and will remain non-partis[illegible]

But on *issues* that affe[illegible] and their families, plus mi[illegible] ers, we have a right—an[illegible]

Horace B. Deets is the AAR

TABLE I

EMPLOYER CONTRIBUTION RATES UNDER KPERS*

A. State Nonschool**

Fiscal Year	Participating Service	Amortization	Group Insurance	Admin.	Total Rate (Rounded)	STATE Employee
1962			--		5.35[a]	4%
1963			--		5.35[a]	4
1964			--		5.35[a]	4
1965	2.311	1.489	--	0.200	4.00	4
1966	2.282	1.518	--	0.200	4.00	4
1967	2.214	1.586	0.50	0.200	4.50	4
1968	1.721	2.079	0.50	0.200	4.50	4
1969	1.634	2.166	0.50	0.200	4.50	4
1970	1.806	3.294	0.50	0.200	5.80	4
1971	3.508	1.892	0.50	0.200	6.10	4
1972	3.613	1.787	0.50	0.200	6.10	4
1973	3.732	1.568	0.50	0.200	6.00	4
1974	4.300	1.400	0.50	0.200	6.40	4
1975	5.000	1.400	0.50	0.200	7.10	4
1976	5.250	1.400	0.60	0.150	7.40	4
1977	5.550	1.000	0.60	0.150	7.30	4
1978	5.005	0.920	0.60	0.175	6.70	4
1979	4.525	0.900	0.60	0.175	6.20	4
1980	4.615	0.810	0.60	0.175	6.20	4
1981	4.025	0.700	0.60	0.175	5.50	4
1982	3.820	0.680	0.60	0.100	5.20[b]	4
1983			0.60	--[d]	4.80[c]	4
1984			0.60	--	4.60[c]	4
1985	2.900	1.100	0.60	--	4.60	4
1986			0.60	--	4.30[c]	4
1987	2.540	0.760	0.60	--	3.90	4
1988	0.700	1.740	0.60	--	3.04[e]	4
1989	0.700	1.740	0.60	--	3.04[e]	4
1990	0.791	1.737	0.60	--	3.10[e]	4
1991	0.864	1.732	0.60	--	3.20[e]	4
1992	0.823	1.896	0.60	--	3.30[e,g]	4%
1993	0.823	1.896	0.60	--	3.30[e]	
Average	2.67	1.51	0.57	--[d]	5.02	

STATE EMPLOYEE RETIREMENT PLAN BENEFITS

State	*Retirement Requirements*	*Benefit Formula*
Alabama	Age 60 + 10 years or 30 years	2.0125% x AFC x service.
Alaska *	Age 60 + 5 years	2% x AFC x first 10 yrs. + 2.25% x AFC x second 10 yrs. + 2.5% x AFC x remaining yrs.
Arkansas	Age 65 + 10 years or 30 years	1.8% x AFC - 1.25% of primary social security x yrs.
California	Age 60 + 5 years	2% x AFC x service
Colorado *	Age 65 + 5 years or 35 years	2.5% x AFC x first 20 yrs. +1.25% x AFC x remaining yrs.
Delaware	Age 62 + 5 yrs. or 30 yrs.	1.67% x AFC x service
Florida	Age 62 + 10 years or 30 years	1.6% x AFC x service
Georgia	Age 65 + 10 years or 30 years	1.5% x AFC x service
Idaho	Age 65 + 5 years	1.67% x AFC x service
Illinois	Age 60 + 8 years or 35 years	1.67% x AFC x first 10 yrs. +1.9% x AFC x next 10 yrs. + 2.1% x AFC x next 10 yrs.
Indiana	Age 65 + 10 years	1.1% x AFC x service
Kentucky	Age 65 + 4 years or 30 years	1.91% x AFC x service
Louisiana *	Age 60 + 10 years or 30 years	2.5% x AFC x service + $300
Maine *	Age 60 + 10 years	2.0% x AFC x service
Michigan	Age 60 + 10 yrs. or 55 + 30 yrs.	1.5% x AFC x service
Minnesota	Age 65 + 5 yrs. or 62 + 30 yrs.	1.5% x AFC x service
Mississippi	Age 65 + 4 yrs. or 30 yrs.	1.75% x AFC x first 30 years; 2% AFC over 30 years
Montana	Age 60 + 5 yrs, 30 yrs. or 65	1.67% x AFC x service
Nevada *	Age 60 + 10 years or 30 years	2.5% x AFC x service
No. Carolina	Age 60 + 25 yrs. or 30 yrs.	1.63% x AFC x service
Ohio *	Age 60 + 5 years or 30 years	2.1% x AFC x first 30 yrs. +2.5% x AFC x remaining yrs.
Oregon	Age 55 + 30 years or Age 58	1.67% x AFC x service
Pennsylvania	Age 60 + 30 years or 35 years	2% x AFC x service
So. Carolina	Age 60 or 30 years	1.82% x AFC x service
Tennessee	Age 60 + 10 years or 30 years	1.5% x AFC +(.25% x AFC above SSIL) x service
Virginia	Age 65 or 30 years	1.5% x AFC x service
Washington	Age 65 + 5 years	2% x AFC x service
Wisconsin	Age 65 + 5 yrs or 57 + 30 yrs.	1.6% x AFC x service

In states which also provide social security coverage, the pension plan's benefit formula ranges from 1.1% to 2.0125%, replacing 33% to 60.4% of average final compensation after 30 years of service. In states which do *not* provide social security coverage (*), the benefit formula ranges from 2% to 2.5%, replacing 60% to 75% of average final compensation after 30 years of service. Average final compensation (AFC) ranges from the average of the two highest consecutive years of service to the average of the five highest consecutive years of service.

Sources: NASRA Survey of Systems, 1989; State of Wisconsin Retirement Research Committee Staff Report No. 79.

Attachment #4-6

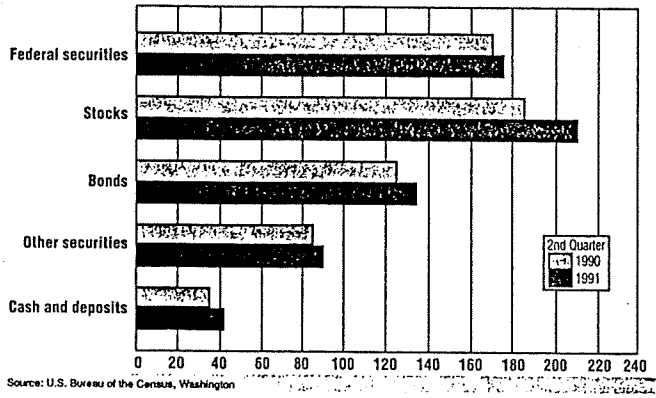
Top 50 public pension funds

	Fund/retirement system	Total assets ($ mils.)	Total managed internally ($ mils.)	Defined benefit asset mix: Stocks	Fixed Income	Other	Investment consultant(s)
1.	California Public Employees	$64,658	$52,903	45%	38%	17%	Wilshire
2.	New York State and Local	50,081	38,271	50	38	12	Callan Associates
3.	New York City	48,876	1,121	53	42	5	Callan; SEI; EAI; Buck; Rogers, Casey
4.	California State Teachers	37,367	20,301	41	47	12	IPC; PCA
5.	New York State Teachers	32,514	29,619	58	31	11	
6.	New Jersey Division of Investment *	31,280	31,280	50	48	2	
7.	Teacher Retirement of Texas	29,477	29,477	46	45	9	Wellington Management
8.	Florida State Board of Administration	25,517	14,526	51	45	4	SEI
9.	Public Employees of Ohio	24,272	23,267	21	60	19	
10.	State of Wisconsin	22,790	21,179	52	41	7	
11.	North Carolina Retirement	20,261	14,051	32	66	2	
12.	Ohio State Teachers	20,200	20,200	35	46	19	Wellington Management; Frank Russell
13.	State of Michigan	20,120	18,910	34	31	35	
14.	Pennsylvania Public School Employees	19,300	3,000	45	50	5	Evaluation Associates
15.	University of California	16,988	16,881	50	45	5	
16.	Washington State Investment Board	15,640	6,082	35	33	32	Frank Russell
17.	Minnesota State Board	15,105	6,305	39	52	9	Richards & Tierney
18.	Virginia Retirement	13,300	718	59	29	12	PCA; Rogers, Casey; Sharpe; RP
19.	Maryland State Retirement	13,200	8,100	36	59	5	
20.	Oregon Public Employees	12,536	3,820	61	22	17	Frank Russell; Wilshire
21.	Teachers Retirement System of Georgia	12,354	7,866	41	55	4	
22.	Pennsylvania State Employees **	11,131	0	48	28	24	Wilshire; Ronald Carp; Pathway Capital
23.	Los Angeles County Employees *	11,091	0	44	40	16	Callan***; The Russell Co.; Chancellor
24.	Colorado Public Employees	10,914	7,780	47	35	18	Mercer
25.	Tennessee Consolidated	10,077	9,968	37	57	6	Wilshire
26.	Retirement Systems of Alabama	9,920	9,920	9	57	34	
27.	Teachers' Retirement System of Illinois	9,888	0	44	27	29	Callan Associates
28.	South Carolina Retirement *	9,386	9,386	0	93	7	Jamison, Eaton & Wood
29.	State of Connecticut Trust Funds	9,360	1,235	49	30	21	TPF&C; PCA
30.	Arizona State Retirement	8,850	0	45	52	3	Mercer
31.	Employees Retirement System of Texas	7,683	7,683	29	70	1	Callan Associates
32.	Massachusetts Teachers and Employees	6,591	3,338	NP	NP	NP	Callan Associates
33.	Iowa Public Employees	5,942	57	35	44	21	Wilshire
34.	Missouri Public Schools System *	5,861	0	18	74	8	DeMarche
35.	Public Employees of Mississippi	5,420	0	40	58	2	SEI
36.	Illinois Municipal	5,318	132	40	29	31	Mercer; IPC
37.	Utah State	4,900	2,700	53	35	12	Callan; SEI
38.	Alaska Retirement Systems *	4,800	2,300	45	47	8	
39.	San Francisco City and County	4,586	2,464	33	55	12	Callan, Townsend; Lewis, Bailey
40.	Employees' Retirement System of Hawaii *	4,520	899	46	32	22	Callan & Wyatt Asset Services
41.	Kentucky Teachers	4,501	1,783	28	52	20	
42.	Teachers' Retirement System of Louisiana	4,292	429	40	50	10	Callan Associates; Becker, Burke
43.	Los Angeles Fire and Police	4,220	108	60	33	7	Wilshire; Wyatt
44.	Illinois State Universities *	4,210	20	57	31	12	Ennis, Knupp
45.	Kansas Public Employees	4,207	0	41	33	26	
46.	Public Employees Retirement of Nevada	4,169	0	40	50	10	Callan Associates
47.	Employees' System of Georgia	4,150	0	NP	NP	NP	
48.	Detroit Retirement Systems *	4,010	0	NP	NP	NP	
49.	Chicago Public School Teachers **	4,000	750	NP	NP	NP	Mercer
50.	Kentucky Retirement	3,900	1,737	55	20	25	Mercer

All data accurate as of Sept. 30, 1991, unless otherwise indicated.
* As of June 30 ** As of Aug. 31 *** Includes Callan Real Estate Services
The following abbreviations were used: IPC (Institutional Property Consultants); PCA (Pension Consulting Alliance); NP (Not provided)
Source: *Pensions & Investments*

Cash and security holdings of selected public pension systems

Survey of 104 systems, constituting about 87% of assets of all public pension funds



Source: U.S. Bureau of the Census, Washington

Public pension plans deal with recession

The economic recession gripping the United States has forced many public pension funds to re-evaluate their strategic plans.

One of the biggest problems facing the pension systems is that financially strapped state governments are eyeing pension money as a short-term, politically inspired way to bring budgets into line, according to Ron Peyton, president and chief executive officer at investment consultant Callan Associates Inc., San Francisco.

Most funds' immediate reaction to the threat of state invasion is to "throw up walls of defense," Mr. Peyton said. "Another reaction is to recognize the needs the state is addressing and see what can be done to satisfy the state yet still serve the pension fund, possibly escaping more severe incursions."

Options that can placate desperate legislators include investing pension money in residential housing within the state or releasing funds for mortgages.

The recession has prompted the Federal Reserve Board to lower interest rates in an attempt to spur more borrowing and spending. That means government pension funds, facing low interest rates on U.S. Treasury bills and other investment vehicles in this country, are hunting for bargains overseas, where investment yields are higher.

Public funds also are interested in foreign markets as a means of enhancing the diversity of their asset mixes, Mr. Peyton said.

In the domestic market, Mr. Peyton noted increasing interest in the stock of small to medium-size companies, based on the assumption that such firms will continue to grow at a faster rate than larger corporations.

—Amy Lamphere

Graphic by John J. Bohorquez

The acceptance of full continuance of net income as a goal for a typical career employee inevitably leads to other questions. How do you measure net income? What period of service represents a full career? How do you treat employee contributions, personal savings, and retiree health benefit costs? What assumptions do you make to compute Social Security replacement ratios? Do you evaluate benefit adequacy for a career employee who retires at age 65 or at age 62?

In 1981, the President's Commission on Pension Policy provided information regarding levels of total retirement income needed to maintain preretirement disposable or net income. Although preretirement net income is difficult to quantify precisely, the Commission's report indicated that full replacement ratios for a single employee range from 75%-85% for lower salary levels, 60%-70% for middle salary levels, and 45%-55% for higher salary levels. Based on the Commission's report and the findings of more recent studies, a replacement ratio of roughly 70%-75% of gross preretirement income would be required to fully maintain the preretirement standard of living of a typical KPERS career employee.

Social Security benefits replace a higher percentage of income for lower-paid than for higher-paid employees. This weighting of the benefit formula in favor of the lower-paid reflects the "social objectives" of the Social Security system. For a career employee who retires at age 65, current Social Security replacement ratios as a percentage of final salary are about 40% for an employee with a $20,000 final salary, 33% at $30,000, 26% at $40,000, and 22% at $50,000. (The current age 62 Social Security replacement ratios are about 32% for a $20,000 final salary, 26% at $30,000, 21% at $40,000, and 18% at $50,000.) If the Social Security replacement ratio for a typical KPERS career employee who retires at age 65 approximates 30%-35%, a KPERS benefit of about 40% of final salary would produce combined retirement benefits that meet the objective of full continuance of preretirement net income.

Annual Notice of Retirees' Rights to Change Tax Withholding

Federal tax laws require the retirement system to provide an annual written notice regarding retirees' rights to change their tax withholding. Retirees may change the amount of tax withheld at any time or elect that no tax be withheld from monthly benefit payments.

Changes in the tax withholding, including elimination of a withholding authorization, may be made by completing IRS form W-4P.

Note: Even if you don't have federal income tax withheld you are liable for payment of federal income tax on the taxable portion of your pension. You may also be subject to tax penalties under the estimated tax payment rule if your payments of estimated tax and withholding, if any, aren't adequate.

Mail tax forms to:

Kansas Public Employees
Retirement System
Capitol Tower - 2nd floor
400 West Eighth Street
Topeka, Kansas 66603-3911

Withholding on pensions is similar to wage withholding. By indicating a marital status and number of allowances on the W-4P form, the amount of withholding will be determined by the tax tables according to your election and the amount of your monthly benefit.

You could also indicate a specific dollar amount you want withheld IN ADDITION TO the tax table withholding amount. Or, you could just indicate a specific dollar amount you want withheld from each benefit.

Withholding deductions currently being made will continue to be deducted until revoked with the W-4P form.

KPERS mailed 1099R statements to the home addresses of all retirees and survivor annuitants receiving a monthly benefit on or before February 1, 1992. Use this statement to file your federal income tax return.

The 1099R shows the amount of gross benefits paid to you during the year, the taxable amount if you retired after July 1, 1986 and the amount, if any, of federal income tax withheld. **You must attach Copy B of your 1099R form to your return if you had federal income tax withheld.** Information about medical insurance premiums of state retirees is also provided.

For those who retired after July 1, 1986 the taxable amount of pension benefits has been calculated in accordance with the exclusion ratio provisions of the Tax Reform Act of 1986. There is an alternate IRS-approved method of calculating the taxable and non-taxable portions of each benefit payment.

Members can use this alternate method - the simplified, safe-harbor rule - to determine the taxable portion of their benefit. Consult the 1040 form instructions for more information.

If you don't receive your 1099R a duplicate may be secured by contacting the retirement system using the address shown in the first column.

1991 Year End Operating Results

Balance at June 30, 1990		**$ 3,750,431,402**
Operating Revenues		
Contributions		
From Members	$ 123,608,209	
From Employers	105,676,734	
Total Contributions	$ 229,284,943	
Investment Income		
Gross Investment Income	$ 292,622,937	
Less: Allowance for Losses on Investments	-153,808,707	
Less: Fees, Expenses Paid Managers, Custodians	-16,590,756	
Net Investment Income	$ 122,223,474	
Total Operating Revenue	$ 351,508,417	
Operating Expenses		
Monthly Retirement Benefits Paid	$ 180,041,274	
Refunds of Contributions to Members	22,086,947	
Death Benefits Paid	6,561,432	
Insurance Premiums & Benefits Paid	23,956,850	
Administrative (KPERS Office) Expenses	3,309,277	
Total Operating Expenses	$ -235,955,780	
Income Over Expenses		$ 115,552,637
Balance at June 30, 1991		**$ 3,865,984,039**

Your Retirement System had total assets of $3.75 billion at June 30, 1990. During the next 12 months, active members contributed more than $123 million to the System while employers contributed more than $105 million.

The Retirement System's investments generated $292 million in income during the fiscal year, including realized and unrealized gains and losses on publicly traded securities. From this amount it is necessary to subtract nearly $154 million. This amount represents the difference between what was originally paid for the direct placement and real estate investments and the current, lower value of those investments. After subtracting management fees and expenses associated with the investments, fiscal year 1991 Net Investment Income totaled more than $122 million, which resulted in Total Operating Revenue of $351,508,417.

The expenses of the Retirement System totaled nearly $236 million in fiscal year 1991. Included were $180 in monthly benefits paid to retired members, $22 million paid to members who withdrew their contributions, more than $6.5 million in death benefits and nearly $24 million in insurance premiums and benefits. The cost of maintaining your Retirement System office totaled a little more than $3.3 million.

Revenues exceeded expenditures by $115,552,637 during the year, resulting in an increase in the System's total assets to $3.86 billion at June 30, 1991. These assets represent the funds available to pay for current and future member benefits.

Attachment #4-9